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Table of Contents.

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ORIGINAL ARTICLES— PAGE.

Rat-Bite Fever: Some Observations, by E. H. DERRICK, M.D., and H. E. BROWN 553
An Unusual Case of Poisoning, with Some Notes on Non-Alkaloidal Organic Substances, by F. W. CARTER, M.B., Ch.B. 558
The Law and the Coroner, by H. RICHARDSON CLARK 565

REPORTS OF CASES—

An Obscure Case of Macrocytic Anæmia, by CYRIL FORTUNE, B.Sc., M.D., B.S., and J. H. COLEBATCH, M.B., B.S. 569

REVIEWS—

Obstetrics and Gynæcology 571
Balneology and Climate 572
Elementary Practical Chemistry 572

LEADING ARTICLES—

House Surgeons and Surgery 573

CURRENT COMMENT— PAGE.

Extract of Suprarenal Cortex in the Treatment of Burns 574
Spontaneous Pneumothorax Simulating an Acute Abdominal Condition 575

ABSTRACTS FROM CURRENT MEDICAL LITERATURE—

Therapeutics 576
Neurology and Psychiatry 576

MEDICAL SOCIETIES—

The Melbourne Pædiatric Society 578

CORRESPONDENCE—

"Ensol" in the Treatment of Cancer 581

BOOKS RECEIVED 582

DIARY FOR THE MONTH 582

MEDICAL APPOINTMENTS VACANT, ETC. 582

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 582

EDITORIAL NOTICES 582

RAT-BITE FEVER: SOME OBSERVATIONS.

By E. H. DERRICK, M.D. (Melbourne),

AND

H. E. BROWN.

(From the Laboratory of Microbiology and Pathology, Department of Health and Home Affairs, Brisbane.)

RAT-BITE FEVER first came to our notice when a guinea-pig developed the disease after injection with liver from a wild rat. This rat has been the only one to pass on the disease out of forty-four tested. It was a female *Rattus norvegicus*, caught alive by Mr. Harry Lambert in a building on the Brisbane River waterfront.

By a coincidence this rat had been under close observation for a fortnight before it was killed. On February 29, 1936, it was injected with a virus being studied in this laboratory and its temperature was taken under chloroform anæsthesia nearly every day for two weeks. The day after the inoculation it gave premature birth to a litter of three, which it promptly ate. The temperature was normal throughout except for the first observation. There were no symptoms either of rat-bite fever or from the injected virus. There was no sign of the conjunctivitis or mangy appearance about the face that is a feature of the late stage of rat-bite fever in rats.

Rat-Bite Fever in Guinea-Pigs.

On the fourteenth day after it was injected the rat was killed and an emulsion of the liver was injected into guinea-pig R 5. This guinea-pig became febrile on the tenth day. The fever continued, except for short intermissions, till a few days before its death on the forty-third day, the maximum temperature being 40·9° C. (Figure I). During the

middle part of the illness there occurred a pronounced inflammation of the vulva with redness and swelling. The animal kept its weight at first, but at the end lost weight rapidly. At the autopsy there were found enlargement of the spleen and congestion of the lungs. Spirilla were not found in the spleen, perhaps because the autopsy could not be made for more than twenty-four hours after death.

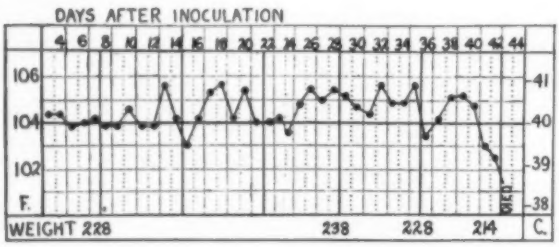

FIGURE I.
Rat-bite fever in guinea-pig R 5, untreated.

On the fourteenth day after guinea-pig R 5 was injected from the rat, 0·8 cubic centimetre of blood was withdrawn from its heart and injected into two male guinea-pigs, R 9 and R 10. From these further passages were made into male guinea-pigs, white mice and a rabbit. All the guinea-pigs developed fever, loss of weight, and inflammation of the scrotum. These features caused a diagnosis of endemic typhus to be made at first; but when the fever still continued after a fortnight and the scrotal reaction too was prolonged and intense, rat-bite fever was suspected. This diagnosis was proved by finding the *Spirillum minus* by dark-ground illumination in the blood, scrotal serum and conjunctival pus of the guinea-pigs and in the blood of the mice. In one guinea-pig the scrotal reaction was so intense that the overlying skin necrosed, forming an ulcer on each side. Spirilla were found in the exudate. This guinea-pig was inoculated intradermally, but did not develop an ulcer at the site of inoculation.

Most of the infected guinea-pigs developed a conjunctivitis, with swollen eyelids and purulent discharge. There was swelling also about the face, with a scaly eruption and a patchy alopecia. The analogous symptoms in the rat are important in transmission of the disease, for it is thought that when a person is bitten by a rat it may be contamination from the eye discharge that introduces the *Spirillum minus* into the wound.[1]

A sign present in these guinea-pigs, which we have not seen previously described, was drooping of the ears. The pinnæ, like the face, developed the scaly eruption. The stiffening then seemed to go out of them and they drooped down. Specific treatment, while it cleared up the scaly condition of the skin, did not restore the pinnæ to their naturally erect position.

Rat-Bite Fever in a Rabbit.

A rabbit was inoculated intraperitoneally with two cubic centimetres of infected guinea-pig's blood. After an incubation period of seven days there began a recurring fever, each paroxysm lasting about one day, with varying intervals between (Figure II). On the eighteenth day conjunctivitis

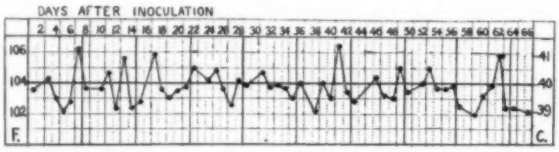

FIGURE II.
Rat-bite fever in a rabbit, untreated.

was noticed, with swelling of the eyelids and, later, swelling of the lips and nose and a scaly eruption, with slight ulceration on the face. The eruption gradually spread until in three months most of the face was scaly and hairless (Figure III). There was also a scaly inflammation on the ears, on the scrotum, and in patches on the back and front. These local inflammations varied in intensity from time to time, on some days the parts becoming very

FIGURE III.
White rabbit three months after infection with rat-bite fever, showing scaly eruption on the face with loss of hair and swelling of lips, nose and eyelids.

swollen and red. The animal ate well and apparently kept its weight, but was lethargic. Spirilla were not demonstrated in the blood by direct observation, but were found in the blood of a white mouse eleven days after its inoculation with the rabbit's blood.

Rabbits are much less susceptible to rat-bite fever than guinea-pigs, and some workers have failed to infect them. The mildness of the disease in our rabbit, in which the paroxysms of fever lasted about one day only, may be contrasted with the severity

of the disease in the guinea-pigs, in which the fever was almost continuous and the result usually fatal. Man is apparently intermediate in susceptibility. In some of the published human cases[2][3] the fever at first was more or less continuous and analogous to the severe guinea-pig type; but as the patient's resistance rose, the bouts of fever became shorter and the intervals longer, as in the rabbit. And in the guinea-pig, treatment changes the fever from the continuous into the recurrent type.

The Spirillum Minus.

The organism causing rat-bite fever, the *Spirillum minus* or *Spirochæta morsus-muris*, may be studied in stained films (Figure IV) or in fresh material by dark-ground illumination (Figure V).

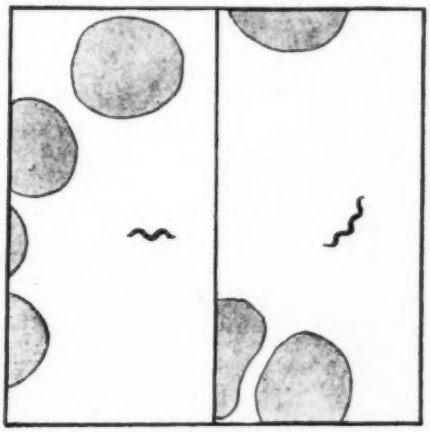

FIGURE IV.
Spirillum minus in mouse blood, stained.

The spirillum will stain with a variety of stains. It is Gram-negative. Following McDermott,[1] we used Adachi's method of fixing the film in osmic acid vapour and then staining overnight with a slightly alkaline Giemsa stain. This gave us good definition of the body of the spirillum, although we have so far been unable to stain the flagella.

When examined with dark-ground illumination in fresh preparations the spirillum is conspicuous by its rapid, darting movements. When it has been exposed to strong light for a short time its movements become sluggish and finally cease. This gives an opportunity to study its shape and the movements of its flagella.

The Laboratory Diagnosis of Rat-Bite Fever.

In making a laboratory diagnosis when a patient is suspected of suffering from rat-bite fever, the first procedure would be the search for the spirilla with dark-ground illumination in the exudate from the sore or in the serum expressed from a papule.

Secondly, white mice should be inoculated with the exudate or with blood taken from the patient during a paroxysm of fever. If present, the spirilla will be readily demonstrated by dark-ground illumination in the blood of the mice seven days later. As mice are naturally subject to infection with a similar or identical spirillum, the blood of those chosen should be examined before injection to exclude this.

A guinea-pig may be inoculated instead, but the spirilla do not become so numerous in this animal as in the mouse. In McDermott's case[1] the diagnosis was made by excising a lymph gland from the child and injecting an emulsion of it into guinea-pigs.

Search for spirilla may be made in films of exudate stained by Giemsa's or Adachi's method.

A positive response to the Wassermann test has some diagnostic value.

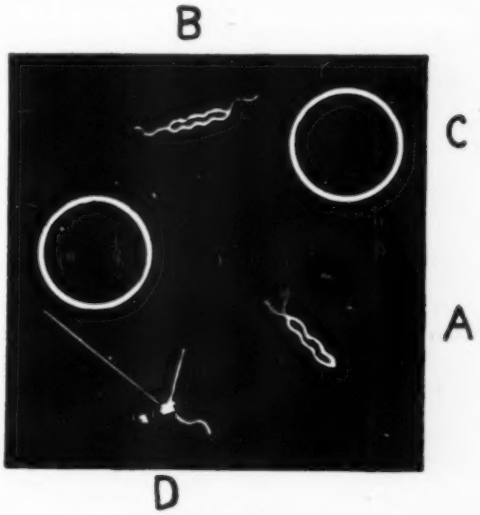

FIGURE V.
Spirillum minus in mouse blood, as seen with dark-ground illumination. The spirilla appear much broader than in stained films. A: Spirillum in motion, being propelled by the posterior flagella. B: Spirillum after exposure to light. Motion has ceased and the flagella have united to form a single curl at each end. C: Red blood corpuscle. D: Platelets with pseudo-spirochætes.

The Wassermann Reaction.

The Wassermann reaction is frequently positive in human and animal cases of rat-bite fever; as also are the other serum reactions (see review by Ripley and Van Sant[2]). Table I gives the results of tests carried out with the sera of our laboratory animals.

TABLE I.

Animal.	Number of Days After Inoculation.	Wassermann Reaction.		Kline Test.
		3 M.H.D.[2]	5 M.H.D.	
Guinea-pig R9[1]	34	++++	++++	
	47	++++	+++	++
Guinea-pig R12	24	++++	++++	
	57	++++	++++	++++
Guinea-pig R13	13	+++	+	
	21	++++	++++	++
Guinea-pig R14	13	++++	++++	
Guinea-pig R14[1]	21	++++	++++	++++
	13	++	+	
Rabbit 8	20	++	+	+++
	34	++++	++++	++++
Mouse 1	34	−		−
Mouse 2	69	−		−
Mouse 3	56	−		−

[1] These tests were performed after treatment was begun.
[2] M.H.D. = Minimum hæmolytic doses of complement.

Treatment of Guinea-Pigs.

Two guinea-pigs—R 9 (270 grammes) and R 14 (305 grammes)—suffering from rat-bite fever, were treated with sulpharsphenamine in doses of 0·002 to 0·006 gramme. In each case the first dose was followed by a striking improvement in temperature, general condition and local inflammation of face and scrotum. Similar improvement, though not always so obvious, followed subsequent doses. The weight began to rise again. As a rule, a relapse, with recurrence both of fever and scrotal inflammation, occurred four to nine days after an injection.

Guinea-pig R 9 was very ill when treatment was begun on the thirtieth day. It had five injections during the next thirty-three days. After the fourth it put on weight rapidly and appeared to be cured. It remained afebrile for twenty-one days, but then the temperature rose to 40·6° C. The animal was killed during the relapse and spirilla were found in the heart blood. It had survived seventy-nine days (Figure VI).

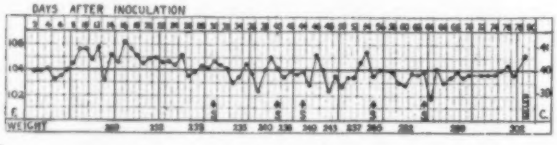

FIGURE VI.
Rat-bite fever in guinea-pig R 9, treated with sulpharsphenamine. Injections represented by S.

Guinea-pig R 14 had five injections, the last on the fifty-second day after infection. It then appeared well, but the fever recurred soon after and has done so at intervals ever since. Scrotal inflammation has been present at times. Mice inoculated with its blood on the one-hundred-and-twelfth day became infected. It has now survived one hundred and twenty-six days, is steadily increasing in weight and is, for the present at least, holding its own with the spirilla.

Untreated guinea-pigs usually die within about two months after inoculation. Six of ours died in 27, 43, 57, 62, 64 and 64 days respectively. This is in contrast with the treated animals. Still better results in treatment would no doubt have been attained if the injections had been more frequent.

In referring to human patients, Ripley and Van Sant state that: "Since recurrences are common when less than three injections are given, a course of from three to six injections seems advisable."

Occurrence of Human Cases.

Human cases of rat-bite fever have been reported from every continent, and the *Spirillum minus* has been found in a proportion of the rats in many places.

Three cases only have been reported in the Australian literature. Muller,[3] in 1918, reported a case in Sydney in a little girl bitten by the family cat. Sinclair,[4] in 1926, described a case after a rat bite in a rat-catcher employed by the Melbourne City Council. Cilento,[5] in 1927, described a case in a white man in New Guinea following a bite by a flying squirrel. The disease is not common, but the reported cases naturally do not represent the full incidence. Dr. K. Winning, Chief Resident Medical Officer of the Royal Alexandra Hospital for Children, Sydney, informs us that there have been two cases in that hospital during the last ten years. We are able to report two more.

Case I.

Dr. D. V. Sheil, of Brisbane, has kindly supplied us with the notes of a case he saw in the Hospital for Sick Children, Brisbane, in 1924. We are greatly indebted to him and to Dr. T. Leckie, the then medical superintendent, and to Dr. D. Cameron, honorary surgeon, for the opportunity to put on record such a striking temperature chart.

The patient, N.H., a boy of seven years, was first seen on May 27, 1924, as an out-patient. He had been bitten by a rat on the right wrist three weeks before this. The wound was not troublesome till a week before he was seen, when a large red swelling gradually developed over the wrist. The swelling was incised on May 31, 1924, without any pus being found. On June 3, 1924, he was admitted to hospital complaining of shivers, anorexia, nausea and pains in the legs. He looked pale and ill, and in addition to the swollen discharging wound on the wrist there were three small red swellings above the right elbow. The temperature rose to 40·8° C. that evening and the pulse rate to 142. The liver and spleen were palpable.

A few days later he was much better. The temperature and pulse rate fell to normal, the swelling of the wrist subsided and the discharge from the wound ceased; but before long the temperature rose again and slightly tender nodules appeared on the left forearm, the right leg and thigh and the right side.

The further course of the illness was a series of relapses, when the temperature rose and the symptoms recurred, alternating with intermissions during which the symptoms abated (Figure VII). At various times

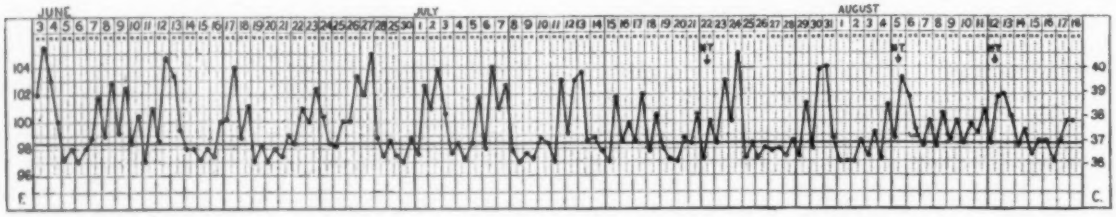

FIGURE VII.
Rat-bite fever in patient N.H. "Neotrépol" injections at N.T.

raised red blotches appeared on the face and elsewhere, subsiding quickly; at one time one side of the face swelled up, at another the left eye became sore. For a time there was numbness in the lower limbs. A systolic murmur could be heard at the apex of the heart throughout the

illness, and at one time a presystolic murmur. The wound on the wrist had healed by June 21, 1924.

Many pathological investigations were made, but the diagnosis was for a time in doubt. Eventually it was made from the characteristic appearance of the temperature chart, together with the history of a rat bite and the otherwise negative findings. It is of interest that the Wassermann test, carried out on July 18, 1924, was found to give a strongly positive reaction.

Treatment was instituted with "Neotrépol", of which three injections were given. This drug, a suspension of metallic bismuth, had just before been introduced by Levaditi with great praise, and this fact, with the convenience of the intramuscular route, probably explains its choice in preference to an arsphenamine preparation, although, in the light of later knowledge, the latter would have given better results. Potassium iodide and other drugs were given by mouth. There appears to have been some improvement from the treatment, but at this interesting stage the parents decided to take the child home.

He had no further specific treatment. The bouts of fever recurred about once a week for about five months after he left hospital, gradually easing off. The blotches continued to come and go at various sites for six years. Since then he has been perfectly well, the only remaining sequel of the illness being a slight stutter.

Case II.

We are indebted to Dr. T. Leckie, now of Ingham, North Queensland, for the notes of the following case.

The patient, B.H., was a boy aged four years. He was seen at his home on January 24, 1936, the complaint being sore fingers and very high fever. He had had a high fever on each of the two preceding days, but appeared well in the mornings. The fever rose suddenly.

Two weeks before the household cat had brought a rat up the back stairs and laid it on the kitchen floor. The rat appeared to be dead. The boy went to pick it up by the tail and it turned and bit the middle finger of his right hand at the tip. The wound healed readily in four or five days and has remained healed. After an indefinite number of days the finger became reddened and swollen and then followed the feverish attacks described above.

On examination the temperature was 40·2° C., the respirations not unduly increased, the pulse varying greatly. The middle finger of the right hand was red and swollen, most of the swelling appearing to be cutaneous and subcutaneous and not at all suggesting a whitlow. The epitrochlear and axillary glands were slightly enlarged. No other abnormality was discovered to account for the fever.

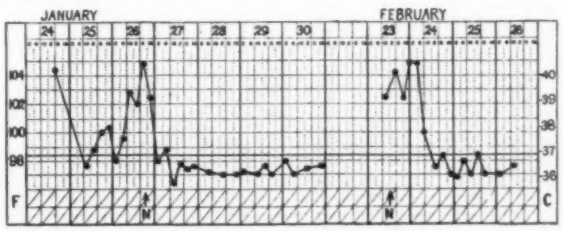

FIGURE VIII.

Rat-bite fever in patient B.H. Neoarsphenamine injections at N.

Since the previous case had been seen it was realized that the type of inflammation was the same in both, and a provisional diagnosis of rat-bite fever was made. The child was admitted to hospital next day, and on January 26, 1936, "Neoarsphenamine", 0·15 gramme, was injected intravenously. The temperature fell to around normal in five hours and remained normal till he was discharged on January 31. (Figure VIII.)

The parents were instructed in the use of the thermometer and were asked to report any rise of temperature. On February 23, 1936, the boy was readmitted to hospital with a rise in temperature at 10 a.m. to 39·1° C. The condition was diagnosed as a relapse of rat-bite fever and he was immediately given 0·15 gramme of "Neoarsphenamine". The temperature rose that night to 40·4° C., then came down rapidly to normal, where it has remained since. He was discharged on February 26, 1936, and has had no further recurrences. He is now (July, 1936) very well.

It is possible that cases of rat-bite fever go unrecognized. The clinical diagnosis is straightforward if there is a history of a bite from a rat or cat or other animal followed by a characteristically recurring fever. Yet in many of the reported cases the history of a bite was obtained only after the diagnosis had been made on other grounds. And the temperature chart is not always characteristic, especially at the beginning, when the bouts of fever may last for some time before the intermissions occur, as in Muller's case and in Ripley and Van Sant's first case. So it is possible that atypical or unrecognized cases of rat-bite fever help to make up the *"Farrago Pyretologica"* (Rolleston) called coastal fever. In a case of protracted fever the history of a bite should be sought and the possibility of rat-bite fever kept in mind.

Summary.

Rat-bite fever was found in a wild rat caught in Brisbane. This is a reminder that the disease is prevalent among rats everywhere and its transmission to man is always liable to occur.

Two cases of rat-bite fever in children are described, one from Brisbane and one from North Queensland.

The possibility of this disease should be kept in mind in cases of protracted fever.

Acknowledgements.

We are indebted to Sir Raphael Cilento, Director-General of Health and Medical Services, for his interest in this work and for permission to publish it. We are grateful also to Dr. G. W. F. Paul, Medical Officer of Health for Brisbane, and his staff, for arranging the supply of rats, and to Mr. W. J. Sanderson, Government Photographer, Department of Agriculture and Stock, for the photograph of the rabbit.

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(1) E. N. McDermott: "Rat-Bite Fever: A Study of the Experimental Disease, with a Critical Review of the Literature", *The Quarterly Journal of Medicine*, Volume XXI, April, 1928, page 433.

(2) H. S. Ripley and H. M. Van Sant: "Rat-Bite Fever Acquired from a Dog", *The Journal of the American Medical Association*, Volume CII, June 9, 1934, page 1917.

(3) O. R. P. Muller: "A Case of Rat-Bite Fever in Sydney", THE MEDICAL JOURNAL OF AUSTRALIA, June 29, 1918, page 531.

(4) T. W. Sinclair: "Probable Case of Rat-Bite Fever", THE MEDICAL JOURNAL OF AUSTRALIA, July 24, 1926, page 111.

(5) R. W. Cilento: "Rat-Bite Fever in New Guinea", THE MEDICAL JOURNAL OF AUSTRALIA, August 6, 1927, page 191.

AN UNUSUAL CASE OF POISONING, WITH SOME NOTES ON NON-ALKALOIDAL ORGANIC SUBSTANCES.[1]

By F. W. CARTER, M.B., Ch.B. (Aberdeen),
Perth.

BEFORE discussing shortly some non-alkaloidal organic substances, I wish to refer to an unusual case of poisoning.

ABOUT 10.30 a.m. on March 31, 1936, Mrs. R., aged twenty-nine years, a delicate *multipara* who had four children, took a dose of what she believed to be mutton bird oil, a remedy given her by her mother and purchased by the latter from a firm of wholesale chemists in the city a few days before. It was the second bottle of the stuff given to her, and on taking it into her mouth she noticed that it differed from the first in both taste and smell. She spat most of it out and was of the opinion that she could not have taken more than half a teaspoonful of the dessertspoonful poured out. She immediately washed her mouth out with water and went on with her housework. She stated that she did not feel very well, but was able to do some washing for the children before getting dinner for the family. When her husband and children came in shortly after 12 o'clock, one of them drew the attention of the husband to her face, which was pale, and to her lips, which were livid. The mother then told her husband of the episide of the mutton bird oil, and it was suggested that she visit her doctor during his two o'clock to three o'clock consulting hour. Shortly afterwards the woman collapsed and I was sent for on urgent summons.

On my arrival about fifteen minutes later the patient was found lying on her bed. She was pale and her lips were cyanosed. The hands and feet were cold and clammy and the nails blue. The pulse rate was 100 per minute and the temperature was subnormal; respiration was slow and shallow. The breath smelled strongly of bitter almonds. The patient complained of severe frontal headache, but both she and her husband asserted that her condition was much improved and were rather apologetic about the urgent call.

Inspection of the bottle from which the dose had been taken disclosed it to be of about one pint capacity, of the size and style usually associated with a pound of fluid cascara or one of the tinctures. It bore no indication, either by shape or label, that its contents were of a poisonous nature. It was nearly full of an oily liquid smelling strongly of bitter almonds. It bore two labels; one was a small square bearing the intimation that on return of the bottle an amount of sixpence would be refunded; the other was the usual square label of large size bearing the name of the supplying house with the name of the contents printed across the centre. On the smaller label was written in pencil: "Oil of Mirbane"; and the same words were printed on the larger label. The patient said that the former bottle had been labelled "Mutton Bird Oil" on the smaller and "Oil of Mirbane" on the larger label.

Because of her improved condition I merely gave her a pint of sodium bicarbonate solution, which was promptly vomited, and treated her for shock, hot water bottles being placed around her and blankets piled on. The condition was regarded as one of prussic acid poisoning, from which the patient was recovering and from which all danger had been eliminated by effluxion of time and the smallness of the dose.

Investigation at a nearby pharmacy into the composition of oil of mirbane disclosed the name to be one of the synonyms of nitrobenzol or nitrobenzene, a highly poisonous organic compound occurring in the synthesis of aniline, characterized by delayed action and a very high mortality rate. I immediately returned to the home of the patient and, much to her disgust administered strychnine hypodermically, her condition in the meantime being unaltered.

At three o'clock the same afternoon it was decided to play for safety, and with a consultant the various possibilities were discussed and the patient was reassured. She was still feeling well.

Between seven and eight o'clock in the evening she suddenly became intensely ill. The pulse rate was 160 and the temperature 37·8° C. (100° F.). Respiration was extremely slow and shallow—about 12 to 16 per minute; cyanosis was extreme, so that the nails of her hands and feet might have been described as being black. The skin had assumed a leaden hue. The extremities were cold and clammy. She was restless and anxious, and complained of nausea and headache. The systolic blood pressure had fallen from 120 millimetres of mercury in the afternoon to 100 millimetres; it later went as low as 80 millimetres. She was removed to hospital.

Because of the depression of respiration it was considered good treatment to give "Carbogen"; and this was done continuously for about six hours. Strychnine and digitalin were given hypodermically and later "Coramine" intravenously with definite, though slight, improvement. The patient was quite conscious; in fact, she remained so throughout, except for the few minutes involved in a convulsion which came on suddenly about midnight and which appeared to presage the end. At this stage blood transfusion was started, with her father as donor, and from then on she slowly improved, so that at about 3 a.m. it was considered safe to leave her with a fair chance of recovery, oxygen being now given in lieu of "Carbogen". She remained relatively cyanosed for over a week, and on the third day developed jaundice, which, in view of its sinister history in other cases, occasioned misgiving. For this calcium gluconate was given by mouth and it slowly cleared up. Later, anæmia became a feature and with it attacks of paroxysmal tachycardia, which occurred even while the patient was resting in bed. She was given one of the liver preparations with iron, plenty of nourishing food and sunlight; a slow return of appetite followed. With a return to activity the heels became painful, and although great care had been exercised in the prevention of pressure during her stay in bed they became pale and slightly anæsthetic; necrosis, however, did not take place, and the condition passed off after a few weeks. Headache and insomnia persisted for three weeks.

A differential blood count done on May 6—about five weeks after the poisoning—showed the following:

Red cells, per cubic millimetre	4,140,000
Hæmoglobin value	79%
Colour index	0·96
Leucocytes, per cubic millimetre	2,600
Polymorphonuclear cells	52%
Lymphocytes	39%
Monocytes	6%
Eosinophile cells	1%
Basophile cells	2%

You will see that there was a pronounced leucopenia and a slight hæmoglobin deficiency. Although the leucopenia was not of the agranular type, it was considered wise to take steps to restore the efficiency of the blood-forming function of the bone marrow, particularly in view of the continued lassitude on even slight exertion, accompanied by breathlessness *et cetera*.

In comparatively recent literature the value of pentose nucleotide in the treatment of agranulocytosis has been demonstrated convincingly. Its action in stimulating leucocyte production is based on the known fact that, in the presence of disintegrated cells of the same type, stimulation of their immature precursors in the bone marrow does take place. Jackson prepared such a preparation of adenine and guanine and it is now marketed

[1] Read at a meeting of the Western Australian Branch of the British Medical Association on June 17, 1936.

under the trade name of "Pentose Nucleotide K.G. 96". Menley and James Limited, of London, issue it in 10 cubic centimetre bottles under the name of "Pentnucleotide", and a small supply was secured from their Sydney agents. Before its arrival a second count was made, on May 20, 1936, when the red cell count had improved to 4,350,000 per cubic millimetre; there was still slight hæmoglobin deficiency, but the leucocyte count had improved to 3,400 per cubic millimetre, with polymorphonuclear cells at 57% and lymphocytes at 43%.

Treatment was begun on May 22, when the patient received two 10 cubic centimetre injections into a muscle. Thereafter she was given intramuscularly one ampoule of 10 cubic centimetres every day, with no reaction as regards discomfort or swelling. Her condition in the meantime was improving and she had now taken over the care of her home and family, with, of course, help.

Her last differential blood count was done on June 3—four days after the last of the 10 injections of "Pentnucleotide" had been given—and the result was as follows:

Red cells, per cubic millimetre	4,340,000
Hæmoglobin value	84%
Colour index	1·0
Leucocytes, per cubic millimetre	5,800
Polymorphonuclear cells	63%
Lymphocytes	31%
Monocytes	3%
Eosinophile cells	3%

There are many points worthy of note in this most interesting case. I would draw your attention to the latent period between ingestion of the poison and the onset of really serious symptoms—about two hours. This was followed by a further period of apparent inactivity, during which the patient felt that her condition was improving. Then came with appalling suddenness all the indications of an impending tragedy, a tragedy which was averted only by the exercise of extreme measures directed against the ravages that had resulted and towards the replenishing of a badly damaged blood supply. The next interesting feature was the stimulating of respiration by means of "Carbogen". In a respiratory centre depressed to almost vanishing point it seems strange that stimulation was possible, yet a relatively small amount of carbon dioxide increased both rate and amplification of the respiratory excursion. The beneficent effect of transfusion could have been of value only because the poison was no longer in the blood stream; otherwise the transfused blood must have shared the fate of the changed blood of the patient. Possibly the bleeding of the patient to the extent of a pint or more before transfusion might have been sounder practice, but since her blood had somewhat the consistency of melted chocolate, I doubt whether any appreciable quantity could have been removed from her veins. I was rather struck with the immediate improvement resulting from the intravenous injection of "Coramine" in contradistinction to the negative results of the other stimulants used. Brandy was, of course, definitely contraindicated.

I should here like to interpose a composite sort of formula of an antidote which has been calculated to supply that something which should be at hand in case of emergency. It is by no means complete and final (no common antidote could be devised that would be even remotely so), but in those ill-defined conditions—ill-defined in that treatment is more or less confined to the combating of symptoms as they arise, and in the elimination of the poison or in its neutralization—it provides a safe and ready means of alleviating the symptoms and assisting elimination, even though it must be regarded as incomplete. It consists of two solutions. The first is a saturated solution of sulphate of iron, 100 parts. The second solution consists of calcined magnesia, 88 parts, animal charcoal, 40 parts, and water, 100 parts. The two fluids are to be mixed at the moment of being used.

This antidote is said to be ideal for arsenic, zinc salts, digitalis and acids of the ordinary type; it is useful for mercury salts, morphine and strychnine, but is of no use for alkalis, phosphorus, tin salts or hydrocyanic acid. May I issue a warning that when such mixtures, which are designed to neutralize poisons by chemical change, are used, the earliest opportunity should be taken to secure their ejection from the stomach; for it may well be that only the rate of the diffusion of the poison has been affected by the antidote, in which case subsequent digestive action may once again secure the passage of the poison into the blood. Emesis should be secured and the stomach washed out. Finally, it may be found wise for the relief of pain and irritation to leave in the stomach when the tube is withdrawn some simple demulcent, with or without an anodyne.

How did this case of poisoning occur? The mother of the patient had an account with a firm of wholesale chemists by virtue of the fact that her husband was an optician. She went in to get a pound of mutton bird oil for her daughter. The correct preparation was placed in a bottle and the small label was correctly marked, presumably by the assistant who filled it. It was then passed on for finishing and the larger label, bearing the name "Oil of Mirbane" was affixed. On receiving it from her mother, the patient noticed the discrepancy in the labels and with her husband came to the conclusion that oil of mirbane was the technical name for mutton bird oil, and for the next three weeks took a dessertspoonful three times a day. When a second bottle was required, the empty bottle was taken to the warehouse for a refill. Although the mother asserts that she asked for mutton bird oil, the bottle was apparently sent to the drug room with instructions to refill, and this was done—with oil of mirbane. Only the foreign odour, noticed at the moment of taking, and the taste prevented the taking of a full dessertspoonful with almost certain death.

Nitrobenzene.

Nitrobenzene—nitrobenzol, "essence or oil of mirbane", imitation oil of bitter almonds—is used extensively in the manufacture of aniline and of explosives; as a substitute for benzaldehyde (oil

of bitter almonds) in perfumery and flavouring extracts; as an ingredient of shoe dyes, floor polishes, inks *et cetera*. Many severe and fatal cases of poisoning have resulted from its being taken internally when used as a substitute for flavouring agents, or in alcoholic liquors, or in confectionery or pastry. It has also been taken as an abortifacient and as a means of committing suicide. It has been absorbed, sometimes with fatal results, when spilled upon the clothes, or in the wearing of shoes recently dyed with liquids said to contain nitrobenzene. Infants have been poisoned by lying on cloth stamped with ink containing nitrobenzene. Poisoning has resulted from its application to the skin in the treatment of scabies or as a delousing agent, and from its application to a carious tooth instead of oil of cloves. Symptoms of poisoning have resulted from the use of soap—"almond glycerine soap"—scented with nitrobenzene; this is most likely to occur when the soap is used in hot water. Industrial poisoning occurs chiefly in plants making aniline.

Properties.—Nitrobenzene ($C_6H_5NO_2$) is an oily liquid, of a pale yellow colour, having a specific gravity of 1·186 and a boiling point of 205° C. (401° F.). It is insoluble in water and is easily soluble in alcohol. The liquid burns readily with a luminous flame, and has a sweet taste and an odour strongly resembling that of oil of bitter almonds.

The Physiological Action.—The physiological action is complex. It stimulates, then paralyses the central nervous system. It also produces changes in the blood; in fact, is a good example of the "blood poisons". The nature of the changes in the blood are not entirely understood. The blood becomes a chocolate colour and some of the corpuscles are deformed, while others are destroyed. The blood becomes incapable of carrying oxygen and may contain but 1% of oxygen (normal 17%).

Symptoms.—When it is taken internally there is a burning sensation, followed by numbness and tingling, but other symptoms may not occur for from one-quarter to two or three hours. The patient may walk about, eat, and appear quite normal, and then the symptoms begin quite suddenly. The face becomes greyish or bluish white, the lips and nails become purple; the pulse is weak and rapid; vision is disturbed; the gait is unsteady; the breath has the odour of bitter almonds; vomiting, dizziness and headache follow, and finally coma comes on, often with great suddenness. Muscular twitchings and involuntary evacuation of fæces and urine may occur. Well-marked trismus, making the passing of a stomach tube difficult, has been observed a number of times. The temperature falls and respiration is frequently of the Cheyne-Stokes variety. The pupils may be dilated or contracted and do not react to light; there may be nystagmus. The urine is dark and smells of nitrobenzene; it contains a reducing substance. The blood is chocolate-coloured, thick and viscid. Death usually occurs in coma from failure of respiration; sometimes it results from the inspiration of vomited matter.

If the patient survives, his condition may apparently remain serious for several hours, and then recovery occurs rather rapidly. Nausea and vomiting may persist; there is frequently a rise of temperature, sometimes associated with necrotic processes, as in one case of the heels. Jaundice may appear in three or four days and continue until death occurs several days (for example, seventeen) afterwards. Ehrlich found the blood in such a case to be very similar to that of pernicious anæmia.

If the poison is absorbed through the skin or lungs, the symptoms may occur very quickly; the patient may become dizzy and faint and collapse within a few minutes; death may occur within an hour. In less acute cases there are drowsiness, faintness, headache, unsteadiness of gait, the patient acting like a person intoxicated, and stupor; the face is flushed, then cyanotic. There is cardiac irregularity; loss of voluntary power and extreme cyanosis occur. Coma may come on very suddenly with symptoms resembling those of apoplexy, death being preceded by Cheyne-Stokes respiration, and occasionally by convulsions.

Men engaged in industries involving the use of nitrobenzene frequently suffer from a form of chronic poisoning in which languor, somnolence, breathlessness, blueness of the face and sometimes failure of the sight are common symptoms. The urine is of a dark maroon colour and on warming gives the odour of the oil of bitter almonds. Adams describes a very chronic case of poisoning, extending over years, in a woman who used nitrobenzene as a cleansing agent for her hands and clothing; the outstanding symptoms were fatigue, multiple neuritis, extreme indigestion, emaciation and anæmia.

Fatal Dose.—Fifteen drops have proved fatal. In another case twenty-three minims, taken in seven doses during forty-eight hours, caused extremely alarming symptoms. Alcohol seems to favour absorption, so that a dose otherwise not fatal may cause death when taken with alcoholic drinks. On the other hand, patients have recovered, under treatment, from three and a half ounces. From a half to one and a half drachms would probably, in ordinary circumstances, cause death. Smaller doses would probably be fatal under some conditions, while recovery would follow much larger doses under appropriate treatment.

Fatal Period.—Death has taken place within an hour from inhalation or absorption through the skin, and within three hours after the poison had been taken by the mouth. In most cases it has occurred within twenty-four hours, but has been delayed forty-eight hours or even longer. In many cases coma has appeared in from three to five hours, and death has occurred six to eight hours later. In some cases death may be delayed for several (seventeen) days.

Treatment.—If the poison has been taken by mouth the stomach should be washed out with warm

water until the water no longer smells of nitrobenzene; a saline purge may delay further absorption of the poison. Oils, milk and alcohol should not be given. If the poison has resulted from the spilling of the poison on the clothes or from its absorption from shoes *et cetera*, these should be immediately removed and the patient given a bath. In mild cases the patients recover without further treatment. A warm bath with a cold douche has proved useful; also bleeding with saline infusion and oxygen inhalation. In severe cases bleeding and blood transfusion are indicated both by theoretical considerations and experience.

Post Mortem Appearances.—The face appears flushed and the lips livid. The superficial vessels, especially those about the throat and arms, are engorged with blood. The organs have the odour of bitter almonds. This odour may persist for several days, whereas the similar odour due to the presence of hydrocyanic acid passes off in a short time. Drops of nitrobenzene may be found in the stomach and intestines several days after death. Ecchymoses may be found in the œsophagus, stomach and duodenum. The blood is fluid and dark brown, and contains "shadows" of red corpuscles as well as distorted corpuscles.

A case of nitrobenzene poisoning was reported in *The British Medical Journal* of March 25, 1933, by C. L. J. Pratt.

In this case a man who worked in an atmosphere tainted with the odour of nitrobenzene began to feel ill; he was moderately cyanosed and was occasionally sick. One day he suffered severe upper abdominal pain and some surgical condition was diagnosed. At operation the spleen was found to be ruptured; splenectomy was done and the man made a good recovery. Examination of the spleen showed thrombosis of the vessels with consequent necrosis of the capsule and rupture; the organ had the odour of bitter almonds.

Drugs Containing the Benzene Ring.

During the past ten to fifteen years drugs such as amidopyrine and arsphenamine, which contain the benzene ring, have come into wide use, and concurrent with their popularity, and probably dependent on it, has been a widespread and alarming increase in the incidence of the disease known as agranulocytosis, agranulocytic angina, or idiopathic neutropenia.

Idiopathic neutropenia may be defined as a disease characterized by sudden onset, fever and, in most cases, sepsis of greater or less degree (usually oral) and extreme leucopenia with a marked reduction in the percentage of polymorphonuclear cells. It is possible to divide the disease into two main classes: (i) primary neutropenia, in which the ætiology is unknown, and (ii) secondary neutropenia, in which the ætiological agents are evident, for example, infections, toxæmias and depressant drugs.

It is of more than passing interest to note that the benzene nucleus occurs in many drugs enjoying more or less popularity throughout the world; the following are but a few of them: acetanilide, phenacetin, arsphenamines, atoxyl, balsam of Peru, balsam of Tolu, benzoic acid and sodium benzoate, benzaldehyde, brilliant green, "Lysol", creosote, fluorescin, gentian violet, guaiacol, mercurochrome, nitrobenzene, phenol, phenolphthalein, phenylhydrazine hydrochloride, picric acid, proprietary headache powders, pyrogallol, saccharine, salicylates, "Scarlet Red", tannic acid, tannates and synthetic tannin compounds (and so in tea), tar, thymol, vanillin, zinc sulphocarbolate *et cetera*.

In recent years, during which the attention of the profession has been focused on the disease, more than 500 cases have been placed on record, and attention has been drawn to the fact that it has appeared mainly in persons who are apt to take such drugs, such as physicians, chemists, nurses and those directly under the charge of physicians, and to a far greater degree than applies to the rest of the community. But it must be remembered that since many patients take amidopyrine and other similar drugs over lengthy periods without any ill-effect, it must be assumed that those patients who develop agranulocytopenia have either an idiosyncrasy or have abnormal bone marrow which is particularly easily damaged by the drug. Nevertheless, in view of these findings it would be unwise to prescribe to persons with a leucopenia the drug amidopyrine or any drug containing the benzene ring.

I do not regard the foregoing as an indication for the scrapping of what may prove, in the hands of some, a useful remedy, but, as with other drugs of the same group, the balance must be held fairly between its usefulness as a remedy and the seriousness, both as regards the immediate results and possible sequelæ, of such idiosyncrasy. Only under conditions of deliberate watchfulness for allergic reaction, together with absolute control of supply, is one justified in exposing one's patient to the risk involved.

Acetanilide.

Acetanilide is a direct aniline derivative and is given no longer as an official preparation. The dose is 0·12 to 0·3 gramme (two to five grains), but much larger dosage has been advocated. It is by no means free from risk, although death is not common. Toxic symptoms have frequently followed its administration, even in medicinal doses. Judged by reported cases, the lethal dose may be somewhere between 0·3 and 0·66 gramme (five and eleven grains). Its action is the formation of methæmoglobin in the red cell, with consequent oxygen starvation.

Treatment lies in the emptying of the stomach, artificial respiration, and the administration of oxygen. Shock is treated along the usual lines, by warmth *et cetera*. It would seem a logical procedure in cases in which there has been a diminution of the oxygen-carrying function of the blood, to increase the amount of available oxygen over a given period. To this end "Carbogen" has been advocated, its usefulness depending, of course, on the presence of a proportion of hæmoglobin in the

red cells still unfixed by the poison and available for oxygen exchange. In all cases of severity, with little or no oxygen-carrying function remaining, blood transfusion must ultimately be done, preferably preceded by bleeding. Assuming that all traces of the drug have been eliminated from the stomach, 500 to 750 cubic centimetres are usually sufficient to tide the sufferer over that period which must elapse before his natural resources come into operation. This statement is made on the assumption that the drug has but one action: the fixing of the hæmoglobin in the red corpuscle.

Phenacetin.

Phenacetin is a coal tar derivative. Its official dose is 0·3 to 0·6 gramme (five to ten grains). It is said to be free from unpleasant effects, but three doses of 0·48 gramme (eight grains) each caused very alarming symptoms. Cases have been reported in which habitual takers have shown blueness of the lips, tongue, ears and finger tips, fainting and pains in the head, rapid and shallow respirations, partial loss of voice *et cetera*.

It is a drug which we all prescribe freely and one which can be and is repeated as often as the patient may desire.

I recently saw a patient for whom I had ordered 0·48 gramme (eight grains) with the same dose of phenazone and 0·12 gramme (two grains) of caffein citrate nearly twelve months before. She was a nervous wreck, with tremulous hands and cyanosed finger tips and lips; her pulse was weak and the rate was 120 to the minute. She complained of insomnia and constant headache and said that she had become entirely dependent on the powders for rest; she had taken nearly 700 of the powders in less than a year. It took me about two months to wean her off them, and such is the craving engendered by their habitual use that I am still afraid that she may return one day to say that she has given up the fight and gone back to them. I should say that her chances in an intercurrent illness would be nil.

Phenazone.

Phenazone was introduced into medicine for its power of reducing fever. It is a very powerful drug and is dangerous for self-administration. Numbers of unpleasant and even dangerous symptoms arising from its use have been recorded, and several deaths. The dose is 0·3 to 0·6 gramme (five to ten grains), but there is a tendency of late to very much larger dosage, especially in children. It has many non-official combinations, of which pyramidon is a notable one.

Poisoning by phenazone is characterized by extreme collapse, coldness and depression of the heart's action. Methæmoglobin is said not to occur. Treatment is along general lines: warmth, stimulation *et cetera*.

"Novalgin."

"Novalgin" is a product of Bayer and is described as "an antirheumatic without any contraindications, well tolerated and frequently effective where salicylates fail". I have used it alone and in conjunction with sodium salicylate. My results have alternated between very good and very disappointing. Occasionally—in two or three cases over the past twelve months—I have seen a condition bordering on collapse, with clammy skin and slight cyanosis and vomiting, repeated after a subsequent dose and not recurring when the drug was removed from the mixture. I have been unable to find anything in the journals relating to this drug and should be glad to hear of any similar experience occurring to any of the members of this Branch. It obviously belongs to the group of organic compounds likely to produce pathological phenomena such as we attribute to the use of amidopyrine.

Carbon Tetrachloride.

Carbon tetrachloride is recommended in the treatment of hookworm disease; its dose is 2·4 cubic centimetres (forty minims) for adults and 0·12 cubic centimetre (two minims) for each year of age of children up to twenty years; a saline purge is given three hours after ingestion of the drug. It is in common use as a solvent of fats and oils. Its use as a shampoo has been discontinued on account of its dangerous properties, it possessing distinct anæsthetic properties and a depressant action on the respiratory and cardiac centres. Given in therapeutic dosage, it sometimes produces toxic symptoms, such as headache, nausea, vomiting and convulsions. If alcohol or fat is given at the same time, severe toxic symptoms may arise. Death has occurred after the taking of four cubic centimetres by mouth in emulsion of gum arabic.

Treatment.—Treatment consists in immediate removal into the open air with artificial respiration and inhalation of oxygen. Death is very rapid, leaving little time for treatment. *Post mortem* appearances show necrotic changes in the liver and fatty degeneration of kidneys and heart.

The Hypnotic Ureides.

Various hypnotic ureides have been put upon the market from time to time under proprietary names, and medical practitioners may in many instances use one or other of these preparations without being aware of their origin as derivatives of veronal. For example, veronal or barbitone is also sold under the names of "Malonal" and "Hypnogen". Other barbituric derivatives are sold under the names of "Medinal" (veronal sodium), "Luminal" (phenyl ethyl barbituric acid), "Proponal" (dipropyl barbituric acid), "Dial" (diallyl barbituric acid), "Adalin" (bromo-diethyl acetyl urea), "Allional" (pyramidon with iso-propyl-allyl barbituric acid), "Cibalgin" (pyramidon and "Dial"), "Dormalgin" (pyramidon with butyl-bromopropenyl barbituric acid), "Phanodorm" (cyclohexenyl-ethyl barbituric acid), "Theominal" (theodromin and "Luminal"), and veramon (pyramidon and veronal) *et cetera*.

The drugs of this group, of which veronal is the principal member, are widely used as hypnotics, and unfortunately are in common use by the laity without medical supervision. Veronal was first discovered, or synthesized, by Merck in 1903 and was

recognized as the most potent hypnotic which had been discovered up to that time. It was first thought to be very safe, but as a matter of fact, though comparatively safe in small doses, it is dangerous in large doses. These drugs must be classed as habit-forming, for there is in some cases a craving for them if they are taken habitually. It must be admitted, however, that continuous administration under medical supervision for long periods has not been followed by bad effects in great numbers of cases. There are numerous accidents from overdosage taken to relieve sleeplessness, and also a number of suicides. Fortunately, in the greater proportion of cases death does not occur, and therefore the statistics give no precise idea of the number of actual deaths.

Veronal is very slightly soluble in water, whereas its sodium salt, "Medinal", is readily soluble. It is very slowly excreted, and therefore its effect lasts for a considerable time, and with repeated doses there is a tendency to cumulative action. It is excreted as veronal in the urine. Normally it produces a quiet, easy sleep without any after-effects; but in large doses or by accumulation in the blood toxic symptoms may arise, including sickness, headache, giddiness, drowsiness and ataxia. The patient sleeps deeply, rapidly passing into coma. The state of coma may be preceded by a state of excitement, with hallucinations lasting from twenty to thirty minutes. The coma is profound, the muscles are flaccid, the reflexes are abolished, and all sensations are suppressed; Babinski's sign may be present. The face is congested; in early stages it may be red, but becomes more and more cyanotic as the respiratory centre becomes involved. The respirations are slow, deep and stertorous, and in grave cases may be of the Cheyne-Stokes variety. The patient may die from respiratory paralysis or slowly recover. The temperature is often elevated, and it is common after some hours to observe pulmonary congestion or œdema, upon which bronchopneumonia may supervene. The duration of coma varies between a few hours and several days. Incontinence of urine and fæces has been observed, as have oliguria and hæmatoporphyrinuria, indicating fairly severe damage to the epithelial cells of the kidneys. Muscular weakness is marked, and lowering of the blood pressure may persist for some time. Skin rashes are common and are of various types: herpetiform, scarlatiniform *et cetera*.

Chronic Symptoms.—Long-continued use of these drugs may lead to intellectual torpor or mental deficiency, loss of memory, inarticulate or stammering speech, diminution of the reflexes, tremor of the hands, and ataxia. Cutaneous affections are common.

Toxic Dose.—The toxic dose of veronal ranges between one and ten grammes (15 and 150 grains), but at least one case is on record in which the patient recovered from 24 grammes (360 grains). Cardill reports a case in which a woman was given 0·6 gramme (ten grains) of "Luminal" and 3·0 grammes (50 grains) of potassium bromide per day for twelve days. She became stuporose, with double incontinence, her pupils were contracted and inactive, with divergent squint; reflexes were unaffected. Under treatment she eventually recovered. Nicol records a case of coma for twelve hours and complete recovery after a patient took 1·08 grammes (18 grains) of "Dial". Leschke records six cases of suicide by means of "Dial", of which the dose was 2·0 to 2·4 grammes. "Nembutal" has been stated to cause death in doses of 0·45 and 0·4 gramme (7·5 and 6·7 grains) when used as an anæsthetic in patients acutely ill.

Treatment.—Treatment is along general lines. Empty the stomach and wash thoroughly with warm water, leaving a suspension of animal charcoal to absorb any poison which may not have been recovered. Strychnine is given in large doses. Saline solution is given intravenously for elimination purposes. "Coramine" and artificial respiration may be resorted to, but the slow elimination of the drug renders this procedure of little avail. Willcox considers that removal of cerebro-spinal fluid is essential in most cases.

Regarding the treatment of poisoning from barbiturates, an interesting communication from the Paris correspondent of *The Lancet* appears in that paper of June, 1935. He states, *inter alia*:

> The vogue of barbiturate suicides has now become so embarrassing that the Minister of Public Health has asked the Academy of Medicine to give an opinion on the desirability of including certain barbiturates and other drugs among those which cannot be bought without the signature of a doctor . . . . Hitherto legislation and practice have wobbled uncomfortably from one extreme to another. There have been times when you could not buy syrup of Tolu . . . whereas at present you can buy without prescription all you need and more to secure your early demise. As for the barbiturates, whose usefulness in insomnia is unquestionable, the suggestion has been made that they be combined with ipecacuanha in proportions assuring the speedy return of the drug taken in suicidal doses; indeed, a formula for the purpose is said to have been devised already. It is hoped that this expulsive compromise will be adopted, for the public is so accustomed to the purchase of certain popular hypnotics that it would assuredly resent the imposition of medical restrictions at this time of day. After all, poisoning by the barbiturates is fairly easily dealt with in hospital, provided the case is not exceptionally severe.

From the St. Louis Hospital encouraging reports of the treatment of more than 200 cases by strychnine injections have been recorded. It is claimed that the treatment is superior to "Coramine" or anything else as an antidote, but the dosage must be large, as much as 50 milligrammes being given intravenously. The average dosage recommended is between 20 and 50 milligrammes, repeated every half-hour till the desired effect is obtained. The treatment is reinforced by venesection, blood transfusion and, above all, artificial respiration supplemented by the inhalation of "Carbogen". Adrenalized serum, given *per rectum* is also useful. Alcohol is contraindicated.

One of the most disquieting features of the subject with which I have so superficially dealt tonight

is the ease with which these potent and often dangerous drugs can be obtained by the public, a state of affairs for which we must accept a fair measure of responsibility. For years past the profession has been flooded with samples of new remedies, or old remedies with some slight variation of formula and a different name, together with reams of more or less convincing literature. They remain on our desks for a time and then one day we strike a case which just seems to have been made for that little bottle of tablets so long asking for trial. So we generously hand it to the patient with instructions to take one three times a day. The tablets probably put the patient to sleep, sweeten up the urine, or relieve the headache, or stimulate the bowel to its pristine activity; and presently the local pharmacist discovers that he has developed quite an extensive trade in that particular neighbourhood for some foreign substance, which, because it contains no scheduled poison, he is at liberty to supply. And today the public is better informed in the matter of self-medication than ever before in the history of the world.

Drastic action has recently been taken by the British authorities in the matter of amidopyrine and its salts, barbituric acid and its many compounds, and many of the sedative and hypnotic remedies which hitherto were easily accessible to the public. When writing a prescription for these poisons the practitioner must see that it contains the following particulars: (i) his signature, (ii) his address, (iii) the date, (iv) the patient's name and address, (v) the total amount of the medicine, (vi) the dose. These prescriptions may be dispensed once only unless the practitioner states on them that they may be dispensed more than once; if he directs that they may be dispensed a certain number of times or at certain intervals, they may be dispensed only in accordance with the directions. If any of the particulars are omitted it will not be possible for the pharmacist to dispense the prescription.

The handling and sale of poisons in the State of Western Australia are governed by the provisions of the *Pharmacy and Poisons Act*, 1910, an Act which leaves much to be desired and regarding which the Council of the Pharmaceutical Association has been endeavouring for some time to have amendments considered by the government of the day, designed to place the Act on a sounder footing, particularly in regard to the packing and control of poisons and poisonous substances. I am fully alive to the gulf existing between retail quantities of the popular poisons and those poisons and poisonous substances sold in wholesale quantities for industrial purposes; but it is surely reasonable that legislation should be effected whereby poisons, industrial and otherwise, shall be packed in some distinctive type of container which would soon come to be recognized as denoting that the contents were poisonous, and that the word "poison" be conspicuously displayed on any such container. Only thus can we hope to abolish the practice which now pertains of supplying potent chemicals and drugs in containers similar in style and colour to those now associated with harmles galenicals. Had the poison which prompted this paper tonight been in a container of such distinctive shape, and had a poison label been affixed, it is reasonable to believe that there would have been no accident. It is surely something amounting to a scandal that a substance so potent is being sold over the counter without adequate investigation as to the qualifications of the purchaser to handle such poison and without inquiry into the use to which the poison is to be put; but when, in addition, it is not even labelled poison and is placed in a plain bottle it becomes a matter for urgent rectification.

In our own sphere, time does not permit of any detailed discussion of ways and means for (*a*) the confirmation of formulæ, many of them most complex, of all new remedies; (*b*) the checking up of extravagant claims by manufacturers; (*c*) the difficulties associated with cross-testing, biologically, chemically and clinically; and (*d*) the cost to the community of such intensive investigation *et cetera*. There has been a move in the direction of the establishment of a research bureau in the Commonwealth, and it would seem that the time is overripe for some action along these lines to be taken. The matter, however, is of world-wide importance, and only by acting in collaboration with some international body could success be achieved and the cost be kept within reasonable limits.

In the meantime I would urge that this Branch of the British Medical Association extend its strongest support to the Pharmaceutical Society in its effort towards amendment of the *Pharmacy and Poisons Act* in this State.

Acknowledgements.

I would like here to place on record my sincere appreciation of the help given by Mr. Frank Gill and Dr. Troup, Dr. Cohen and Dr. Hayward in an exceedingly worrying case; their ready response to my call for assistance undoubtedly saved the life of the patient, and I thank them.

Addendum.

Since the foregoing was written, namely, June 18, 1936, the following interesting case has come under notice.

A male, aged forty-seven years, complained of incessant headache over a period of eight months. He had had a minor accident in October of last year, resulting in infection of two fingers. For pain he had been given a mixture of aspirin, phenacetin and caffeine, and for eight months had taken two sixteen-ounce bottles a week. He was emotional. Examination failed to disclose any departure from normal, except very definite cyanosis. Blood examination showed the characteristic spectrum of methæmoglobin. With difficulty he has been persuaded to abandon his phenacetin-containing remedy, with the result that the cyanosis is clearing up gradually; his headaches are less frequent and less severe, so that he is now able to sleep through the night without interruption; his confidence is returning, and with it his energy. I feel justified in assuming ultimate recovery from what apparently was a definite case of chronic phenacetin poisoning.

THE LAW AND THE CORONER.[1]

By H. Richardson Clark,
Coroner at Parramatta; Solicitor, Supreme Court, New South Wales.

I assure you, gentlemen, that I feel it a great honour, and it is indeed a pleasure, to address you in an informal way concerning my duty to you as coroner and your duty to me. It throws my mind back many years to the time when I addressed the doctors in Parramatta on the same subject. Although there has been little alteration in the law since then, I feel that there has arisen a new generation of doctors who may not have had the time to devote to a study or perhaps even to a consideration of the subject, which, after all, is extremely simple. Right up to the present, and for many years, doctors have quite frequently asked my advice in regard to their position when a certain class of death has occurred, and I acknowledge with gratitude their confidence and courtesy, as it makes certain a pleasurable contact between us. As that thought is in my mind I shall tell you, so far as I can remember, of the class of case in which I have been consulted.

It will be within your everyday experience that old people falling and sustaining a fracture invariably die quickly thereafter, and the question arises whether you ought to give a certificate of death. The answer is "no", as primarily it is a violent death into which the coroner is bound to inquire, death being really due to the fall, whether accidental or not. Many doctors have inadvertently given certificates in those cases, knowledge of which, however, reaches me through the Registrar of Deaths, whose duty it is to inform me of any registration of a death which is not natural or which ought not to have been certified. As I write, little matters occur to me, and it seems convenient to say: "Do not report to me or sign a certificate that a person has died from 'natural causes'." That really means nothing, as there are so many natural causes of death; say specifically the cause of death. And, again, do not report to me or sign a certificate that a person has died from "heart failure", which means nothing, since we all die from heart failure. State specifically the cause of death.

Difficulty frequently arises when doctors have been called in after death has occurred from a natural cause, such as a cerebral hæmorrhage, or heart disease, or pneumonia (as frequently happens in children who have been unattended for a cold). Can the doctor give a certificate? In the past he could not, as he had not attended the person in life; but that is now altered, as I will presently explain. When going into this subject many years ago, I was astonished to find that there was no legal foundation for a doctor's certificate of death. He was not legally bound to give it and, so far as Acts of Parliament were concerned, it had no legal force. But, as I have said, that has been altered quite recently, and for fear that through demands your work makes upon you, you have not had time to realize it, I shall endeavour to explain the alteration.

On March 30, 1935, Act Number 43 of 1934, passed in December of that year, was proclaimed. It is an Act to amend the registration of births, deaths and marriages. Section 19A provides that the birth of a stillborn child shall be registered; a stillborn child means one of seven months or over not born alive and any child not born alive which measures at least fourteen inches, but excludes any child which has actually breathed. Under Section 27A, doctors must be furnished by the government with printed forms of certificates of causes of death. When the doctor has attended a person in his last illness he must forward at once to the registrar a certificate stating the cause of death, and as well deliver to the tenant of the house or place where death occurred a notice in the form of the tenth schedule to the Act, which merely says: "I hereby give you notice that I have this day signed a medical certificate as to the cause of death of John Brown who died at 16 Edwin Street Croydon on 20th June 1935." This notice is signed by the doctor, who also gives his address and date of signing.

Sudden deaths must be reported by the doctor to the coroner. I shall deal with sudden death first. Take, for instance, "heart disease". You will report that to the coroner. But by a later provision, although you have not attended the deceased, but have seen the body and are satisfied that death was due to a natural cause (this, of course, is purely general and means any one of the specific causes of natural death), you may, with the coroner's approval, issue and sign a certificate of death and also the notice to the tenant of the house. It will be noticed that you must report a "sudden" death to the coroner; but the Act does not say that you cannot give a certificate.

It was in the past left to the coroner to order burial; but now, after reporting to the coroner, you are able to give a certificate for a person whom you have not attended if sudden death is natural. I think this construction is borne out by the eleventh schedule of the Act, which is the form of certificate of cause of death; in a little square is a place which you may initial, stating that you have reported the death to the coroner. If there be no necessity to report, you will take no notice of that little square. It does not mean that you must report, unless death occur suddenly; but when you are doubtful, please report.

In any death of a suspicious nature you are bound to report to the coroner and refrain from giving a certificate of death. I shall not attempt to define a suspicious death, as I think your knowledge on the subject is better than mine and that you are able easily to distinguish a natural death from one which is not or may not be. But fortunately we

[1] Read at a meeting of the Western Suburbs Medical Association, New South Wales, on August 28, 1935. Received for publication on August 18, 1936.

meet with few criminally suspicious deaths; the word "suspicious", however, may include deaths probably due to some accidental violence, such as a fall or injury in a fight, and certainly it would include deaths due to inattention and neglect on the part of those attending the deceased, or inattention at a birth.

The deaths of all patients attended by you during a last illness you may certify. Of course, it is understood that the illness is natural and not due to violence, poison, illegal operation or inattention. But suppose you attend over a period a person suffering from heart disease, and that person, while running for a train one morning, suddenly dies; can you certify that death? Yes, after reporting it to the coroner.

In giving a certificate without consulting the coroner you must bear in mind the distinction between a sudden death and an otherwise natural death. Within my limited knowledge of medical subjects the best example I can give of the latter is as follows. Supposing you are called in for the first time to see a sick baby or older person, and that baby or person is dead on your arrival. You are given a history of cold and cough, and you come to the conclusion that death was due to pneumonia. Well, as you see, that is not a sudden death, since there had been a period of real sickness, and you may, if you wish, give a certificate without consulting the coroner. But, of course, if you think there has been improper neglect in not seeking medical advice, you had better report the death.

Since the recent law authorizing doctors to sign certificates of death when they have not attended deceased persons in life, the question has arisen whether they are competent to sign the forms under the *Cremation Act* for cremation, and whether the coroner, having been informed by the doctor beforehand concerning the certificate, should be called upon to intervene. Certainly not, because I think that when a doctor gives a certificate of death, the matter of cremation is entirely between the Cremation Society and the doctors. The society, however, says: "Oh, we can't cremate because the doctor has not attended the deceased during his last illness, according to the regulations under the *Cremation Act*, but we can cremate if the coroner will give a certificate." That is not so unless the coroner has ordered burial; the way out for the society is to order a *post mortem* examination. So as to make this quite clear to you, you should understand that, according to the regulations, cremation can be effected only when a certificate under the Act has been given by the doctor who attended the deceased during his last illness, when a *post mortem* examination has been made by a doctor expert in pathology appointed by the society, or when the coroner has issued a certificate under the regulations. So you see, solution of the question is quite simple: if no doctor attended the deceased in the last illness, then there can be a *post mortem* examination and the society will cremate the body; but in cases of urgency I have helped the relatives out of a difficulty by giving an order for cremation.

While speaking of *post mortem* examinations it occurs to me to say to you that occasionally when a natural death has occurred and a certificate has been given the doctor applies to me for permission to hold a *post mortem* examination, which, of course, I have no power to give, the matter resting entirely upon the wishes of relatives of the deceased. And again, assuming I have given my order for burial of the body and a *post mortem* examination is desired by the relatives, it can take place without reference at all to the coroner. It is only in cases with which the coroner is dealing and in which he has not authorized burial that interference with the dead body is very wrong, unless undertaken upon his instructions. This subject again reminds me that perhaps some of you are not familiar with the instructions of the Health Department regarding the conduct of a *post mortem* examination. The three cavities, head, chest and abdomen, must always be opened. If you do not do this you will not be paid the standard fee of two guineas. The body should also be identified to you with certainty, and in making your report of the result (for which generally a printed form is provided) you should begin by saying:

> I, ... (stating your full name and place of abode), medical practitioner, do hereby certify as follows: I am a legally qualified medical practitioner carrying on my profession at..... On the.....day of.....19.., I made an internal examination of the dead body of a man identified to me by Constable Jones, of Burwood, as that of John Brown, aged about 50 years. I opened the three cavities, head, chest and abdomen, and upon examination found .....

Then describe the condition of the internal organs and of the brain and any peculiarities in either. State whether there were any external marks of violence or evidence of there having been any. State how long after death the examination was made, where it was made, and the date and hour. Then give what you consider to have been the cause of death.

If you are called upon to make such an examination in case of poisoning you must be thorough and careful and must observe the following directions in removing viscera for examination by the analyst. Remove the stomach and contents, portion of the small intestine and contents, the kidneys, spleen and portion of the liver. When murder is likely, the specimens should be sent in separate jars and the whole of the liver should be sent. Solid organs should never be placed in the same jar as the stomach and intestine.

In suspected poisoning by arsenic or other metallic poison a jar of muscle should be sent as well. In suspected chronic metallic poisoning, for example, by lead, arsenic *et cetera*, you should also send some bone, skin, nails and hair (of this last at least two grammes, preferably five, should be sent), and in all cases wherever possible stomach washes, vomit, urine and similar specimens should be sent

in addition to the others. As regards stomach washes, vomit and urine, the whole quantity, not portion of it, should be sent. Each jar should be carefully sealed and properly labelled. Sealing wax must not come in contact with contents. Zinc plaster should not be used.

Years ago, as coroner, I experienced great trouble over abortions. They were frequent, but nowadays hardly any occur, and it is rarely that one strikes me as not all it should be. There are many natural and accidental miscarriages; that is where your difficulty arises. The well-known confidence which we all applaud and rely upon between patient and doctor ought not to be disturbed, and I know how difficult it must be for you to feel that you should report a case of miscarriage which may or may not have been due to accident. I think it wise to advise you to report any case of death due to miscarriage when you are doubtful of the cause. Some have been so reported to me and I have not held an inquest; you may be sure that I am not looking for wrongdoing and that I am glad to accept a reasonable explanation. This subject is difficult to generalize upon, but I think you should report any instance of what you believe to be a miscarriage brought about by illegal means or probably due to wrongdoing.

I may say a word here as to the privilege of a doctor not to disclose any fact which may come to his knowledge. It seems plain that he has no such privilege, and in court may be legally compelled to divulge all that has passed between him and his patient.

I will read you the statement made in July, 1925, by a well-known English judge, Mr. Justice McCardie:

> The medical profession normally was under the duty of keeping inviolate the secret knowledge that they might gain from treating their patients, and indeed might become liable in a civil action for damages if, without lawful excuse, the duty of confidence was broken; but in a court of law a doctor had no privilege similar to that of a solicitor or other legal adviser, and he was not privileged from compulsory disclosure of communications, however confidential. A further point arose in the case as to whether the doctors were in a specially privileged position owing to the fact that they were acting in a department under the control of the Ministry of Health, through the local committee. In his view there was nothing in the regulations or in any regulations he had heard of which saved the doctor from the obligation of disclosing, if ordered to do so by the court, all the information he might have of the facts he had gained while acting under the regulations.

Earlier in the case the judge had intimated that if the documents required were not produced by the doctor he would commit him for contempt of court.

I think you will find the police in my district of good class, very reasonable, courteous and tactful; and if any of them go to you for information, they go at my request, impliedly or specifically. The final determination of what will be done and the evidence you will be called upon to give will rest with me as coroner, and I have already said that my idea of the administration of the law on this subject is not to disturb, unless quite necessary, the confidence that we know exists between patient and doctor.

You may like to know the extent of my coronial district. My official designation is "Coroner at Parramatta", which has obtained for very many years, but is now a misnomer, as I am more a suburban coroner than anything else. My area extends from Lewisham Bridge to Blacktown, from Hurlstone Park to George's River and East Hills, and then sweeps right across from south to north, including Haberfield, Woolwich, Hunter's Hill, all the northern line to the Hawkesbury River, and also includes all the hills district of Parramatta. So it is easy to imagine that there is always something happening.

I expect you have been very interested lately in reading about what we may call the "shark arm" case and the jurisdiction of the coroner to hold an inquest. He cannot hold an inquest unless there is a dead body; and he must view that dead body, which is technically part of the evidence, as in the old days it lay in court all through the inquest. He ought to look at it thoroughly for marks of violence or anything showing the cause of death, but I am afraid that is not always done. The law originated in the time of King Alfred, when, I suppose, medical science hardly existed, so that the coroner and his jury had to find out for themselves the cause of death. But nowadays we know that coroners are only too glad to leave that question to the doctor. Therefore, in making a report on a cause of death when you have not attended the deceased, you should examine the body thoroughly for marks of violence.

You are aware that in most country towns there is a government medical officer, who is given preference in regard to the coroner's work. In Parramatta there is a government medical officer, but only for that town. So that in the large area from, we shall say, Lidcombe to Lewisham, I must rely upon any doctor resident there; and for some years I have found it to be of convenience and benefit to us all to select one gentleman to do the work. Dr. Hetherington acted for me for some years, and now my very good friend, Dr. Brooks. The gentleman selected, in continually dealing with deaths under my jurisdiction, becomes very proficient and also comes to know what is required by me and the police. Moreover, the police know that they must call in that gentleman, which is much better than their calling any doctor; this, however, they have no power to do, as the privilege is entirely that of the coroner. Believe me, this system is highly advantageous. I have in my mind a particular case in which the fact of the police not consulting me caused great difficulty. The selection of one doctor to do the work also enables him to advise any colleague in matters pertaining to the law.

The coroner must not intervene of his own motion in any death without having information or a

request from the police. But he must hold an inquest upon a person dying a violent or unnatural death, that is, when casualty causes death, when persons are found dead or die in gaols, asylums or lock-ups, when suicide occurs, when prisoners are executed in gaols, when deaths occur from accident in factories or in mines, and, lastly, when deaths occur in fires. It is the duty of a coroner to hold an inquest unless he has obtained information sufficient to satisfy a reasonable mind that death arose from illness or from some other cause rendering an inquest unnecessary. There ought to be reasonable suspicion that death was due to violent and unnatural means.

It is painful to us all when the death of a patient occurs under an anæsthetic. When summing up the evidence I generally say that it should be realized that a doctor hates to lose a patient, especially in circumstances such that it may be inferred death was due to some fault of his. These deaths occur only occasionally, and usually when the anæsthetic has been administered by a doctor of very limited experience. I feel sure that the administration of an anæsthetic should be conducted with as much art and skill as a surgical operation, and, naturally, by an experienced man; that is the opinion of Dr. J. V. Duhig, of Brisbane, stated in a lecture. When I say that deaths from anæsthetics have arisen when they have been administered by quite young doctors, I have in mind several which have recently occurred. In one public hospital for the last twenty years there had not been one such death; but two occurred not so long ago, when anæsthesia was induced by young men. In another instance in which an anæsthetic had been given by one young man and not so long afterwards by another young man in the same hospital to the same person, death promptly occurred. I refer you to the address of Dr. Duhig, of Brisbane, in which he expressed resentment that coroners should hold inquests on deaths from anæsthesia, because, he said, surely it was recognized that anæsthetics were always administered by experienced doctors—which they are not! I can quite understand that there are specialists in anæsthetics as well as in surgery and other branches of your profession, and a specialist in anæsthetics would have an almost psychic knowledge of the state of his patient and would take measures accordingly; whereas a young man of six months' or twelve months' standing as a doctor could not and ought not to be expected to administer anæsthetics with the same safety to the patient as the specialist. I am quite aware, of course, that many operations must take place unexpectedly. I have known of deaths after quite simple operations on young and old.

Cremation is quite modern and is a subject upon which, I am sure you will all agree, great care and caution are to be observed. I give a great number of orders for cremation of people who die in such circumstances that the death comes under my jurisdiction, and I never sign an order without thorough police investigation and consent, nor without reports from two doctors (although I can do it on one report); and sometimes I order an internal examination. The secretary of the Cremation Society told me the other day that up to the present 10,000 people have been cremated under its auspices. Everybody is getting used to cremation, and you and I are used to authorizing it; thus, as the pitcher goes often to the well, it is possible that one of these days a cremation may be authorized which some of us may regret. I am very cautious, and I recommend you to be so too. I once authorized cremation on the report of one doctor when suicide was suspected. I have been sorry ever since, because I do not think it was suicide. If you are called in to confirm the opinion of another doctor, do not take everything as read—be sure. We are all likely to be misled or deceived.

Generally speaking, I authorize burial without knowing anything about cremation; then the relatives wish to cremate the body. I have already the opinion of one doctor. Then I am asked to sign an order for cremation. I say: "Very well, I will send another doctor, whom you must pay, to advise me further as to cause of death before I will authorize cremation." If I should call you, make a careful examination and give me a detailed report in writing as to whether the appearance of the body is consistent with the apparent cause of death, of your conference with the doctor who had already advised me, as to whether there are any marks of violence on the body, whether there is any fact which appears or occurs to you as suspicious, and whether you agree that cremation is proper.

Cases arise in which deaths have occurred from violence, miscarriage, accident or some other cause which may not be natural, but of which the person before death might give information pointing to negative criminality, negligence or fault in some other person. If no such information is given and death ensues, it is sometimes difficult or impossible satisfactorily to decide the question. So I advise you first of all, for instance, in these suspicious miscarriage cases, if you think death will occur, to let the police know, so that they may see the patient and, if necessary, have the dying deposition taken. The same applies in any other violent death, for two reasons: if some other person be criminally responsible, the dying deposition of the patient will become evidence on death; if, however, the deposition or any statement exculpates everybody and shows the patient to be responsible for the act causing death, much trouble is saved. I imagine that these cases are most likely to occur in hospitals, to which people who have met with accidents are most frequently taken. Take, for instance, a person run down by a car. Of course, generally he dies unconscious; but in some cases this is not so, and a dying deposition or statement will be valuable. In any case of accident, as of a workman at work, or an old lady falling over at home, or indeed in any accident when the patient is likely to die, it is a good plan to let the police know, so that the person can be interviewed.

The fee for a *post mortem* examination is two guineas, and it must appear clearly on the evidence that the three cavities mentioned have been opened. But in the circular from the Attorney-General to coroners he says that a *post mortem* examination is unnecessary merely to determine the precise cause of death if there is nothing suspicious about it (that is, if it is not a violent death, such as due to poisoning, a blow, starvation, suffocation, strangulation or any other unnatural cause).

Of course, apart from this there are many cases which appear strange and must be properly examined, such as one I had the other day from Burwood. A young woman, apparently healthy, became suddenly ill, vomited and died; the cause was mysterious, but there was nothing to show that it was wicked. The *post mortem* examination proved that death was due to cerebral hæmorrhage.

Don't be late for an inquest. Don't forget you ever saw the deceased. Don't forget his name. Don't forget where he lived. Don't forget when you last attended him. Don't forget his ailment. Don't forget when he died. Don't write me a letter which no one can read.

Sometimes interesting questions of medical jurisprudence arise in the course of my inquests. One particularly I remember some years ago, when a little boy, running about the yard in his home, fell and cut the femoral vein, which naturally caused profuse bleeding. The parents called personally on a doctor and asked him to attend, but for some unsatisfactory reason he declined, and in the absence of medical attention the child died. So the question arose whether the doctor had committed any offence against the law. After mature consideration and searching the law books, I decided that he was not liable for any offence whatever. He was not the medical attendant of the child and had never previously attended him, so that there was no obligation whatever upon the doctor to go out of his house to the child—that is, I mean, legal obligation. I could not find in our English law books any case on all fours with this, or even approaching the same set of circumstances, but, strange to say, I found the very case in an American law report. A doctor, even on tender of his fee, had refused to attend a person who was very ill, whom he had never attended before, and who died. The relatives sued the doctor for damages, but were defeated upon the ground that there was no contract compelling the doctor to attend the patient.

The Acts dealing with the medical profession are preventive and not compulsive measures. When a doctor qualifies, the State does not require and he does not engage that he will practise at all or on other terms than he may choose to accept. Therefore the conduct of a doctor to a patient can come in question only when there is a contract expressed or implied between them. For instance, if a doctor attends a patient today and refuses to attend him tomorrow, and the patient dies because of the lack of medical attention, the doctor may in a proper case be liable civilly and probably criminally, because there is a contract.

That is, of course, the legal side; but all our doctors recognize the moral side. They well know that skill is a gift by the cultivation and exercise of which it is honest and honourable to live and to prosper, but which it is neither honest nor honourable to withhold from suffering fellow creatures in their time of need. This skill is part of the common heritage of mankind and, by every principle of charity, those who are richly endowed with it are bound to give out of their abundance. Duty arises from contract or implication of law. A doctor attending a patient has a duty to use ordinary skill. A man steals your money and is sent to gaol, because it is his duty not to steal. A man, a Christian Scientist, has a wife or child ill with diphtheria and treats that wife or child according to the principles of his belief; nevertheless, if the wife or child die, he is liable to be sent to gaol for manslaughter, because it was his duty to call a doctor. The law also says that a parent who neglects his child by not calling medical aid is liable to be punished. Similarly it is a doctor's duty to attend without question any of the members of a lodge, and as well to continue to attend a patient under his care.

Here is a delightful picture of the kindly, humane and generous doctor given by his wife:

> We are not rich in the bank, but we have always prospered and we have quite enough. I never walk out with my husband but I hear the people bless him. I never go into a house of any degree but I hear his praises or see them in grateful eyes. I never lie down at night but I know that in the course of that day he has alleviated pain and soothed some fellow creature in the time of need. I know that from the beds of those who were past recovery thanks have often gone up in the last hour for his patient administration. Is not this to be rich?

Reports of Cases.

AN OBSCURE CASE OF MACROCYTIC ANÆMIA.

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and

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W.F., a saddler, aged sixty years, was first seen as an out-patient, complaining of rectal pain on defæcation over a period of six years. An operation for anal fissure was advised. On admission (May 8, 1935) he gave a three or four months' history of pains in the fingers and toes, increasing dyspnœa on exertion, weakness, loss of energy, dizziness, inability to concentrate, insomnia, and pallor. He had a chronic cough with sputum, which was becoming worse. There was slight loss of appetite, but no soreness of the tongue, nausea, indigestion or melena. It was thought that he was losing weight; the bowels were sometimes constipated, sometimes he had diarrhœa; micturition was normal. No drugs had been taken recently and there was no history of contact with any chemical poison. He had had "yellow jaundice" in 1900. His family history was good.

The patient was well built; he had a pale, puffy appearance, and the skin on the abdomen was lemon tinted. His temperature was 37·1° C. (98·8° F.), his pulse rate was 100, and his respiratory rate was 24 to the minute. His systolic blood pressure was 120 and his diastolic pressure was 70 millimetres of mercury. His teeth were artificial; his tongue was smooth, moist and pale; his throat was normal. His heart was slightly dilated, a hæmic murmur being present. Generalized emphysema of the lungs was present with a few scattered rhonchi; fine râles were audible at each base. The liver was enlarged to 5·0 centimetres (two inches) below the right costal margin. The spleen was not palpable. The lymphatic glands were not enlarged. The nervous system was normal (pallæsthesia was not tested). The patient had an irregular anal fissure one inch long, which was not bleeding. The urine was acid and contained neither sugar nor albumin.

On May 2, 1935, the erythrocytes numbered 1,340,000 per cubic millimetre; the hæmoglobin value was 36% (Sahli); the colour index was 1·34; leucocytes numbered 1,000 per cubic millimetre; polymorphonuclear cells were 14%, lymphocytes 86%. The erythrocytes were well hæmoglobinized and showed anisocytosis, but there was no evidence of the definite macrocytosis and poikilocytosis expected in a pernicious anæmia with so low a count. The platelets were rather scanty; no nucleated red cells were seen, but some polychromasia was present. At the time of the patient's admission to hospital (see Table I) the erythrocytes were down to 990,000 per cubic millimetre and the hæmoglobin to 27%; the reticulocyte count was less than 0·5%.

Treatment was commenced with intramuscular injections of an active liver extract, four cubic centimetres daily, in addition to cooked liver by mouth. Following the publication of the results of massive dosage by Gardner and Wood,[1] daily injections were discontinued after two days and a single dose of 16 cubic centimetres was given. However, reticulocyte counts failed to rise above 1% and the hæmoglobin value fell to 22% and remained at that level for some time.

No change in the patient's condition was apparent after ten days, so "Marmite" (30 grammes daily) and a blood transfusion were given. Meanwhile the erythrocytes showed a further decrease in number, and it required a second transfusion two days later to raise the count above 1,000,000. A fairly severe rigor followed each transfusion.

At this stage the leucocyte count was low again (1,000 per cubic millimetre) and attempts to raise it were made by giving intramuscular injections of sodium nucleinate and of a "leucocyte cream" prepared as described by Strumia.[2] A migration of immature granulocytes into the circulation followed, but apparently was of no avail. Two days later the soft palate and fauces were covered with thrush-like patches from which were obtained staphylococci and a fungus resembling *Monilia albicans*. A further 20 cubic centimetres of liver extract were given without any effect on the blood picture. Despite intensive local treatment the throat condition developed into a true angina, and was followed by hypostatic pneumonia from which death occurred on May 29, 1935. Throughout the illness there was an intermittent pyrexia to 38·3° to 38·9° C. (101° and 102° F.), subsiding a little during the second week and rising to 40° C. (104° F.) in the last two days when signs of pulmonary consolidation appeared.

A few additional investigations were carried out after the first week. The blood did not react to the Wassermann test. The Van den Bergh test gave a weak positive indirect reaction. The blood urea content was 51 milligrammes per 100 cubic centimetres. As explained below, a test meal was not performed, but analysis of the gastric contents withdrawn by puncture nine hours after death revealed no free hydrochloric acid.

Post mortem the right femur was split open longitudinally and the marrow was found to be pink throughout the proximal half and yellowish-white distally. The marrow of one of the larger ribs was uniformly dark red, and that of a lumbar vertebra was only slightly paler. The gastric mucosa was atrophic and almost devoid of rugæ. Examination of the lungs revealed left basal consolidation with some collapse and right basal consolidation with fibrinous pleurisy and 300 cubic centimetres of brownish effusion. The heart was small, with a few epicardial petechiæ; slight coronary and aortic atheroma was present. The liver was slightly enlarged, pale and fibrotic. The spleen was enlarged; it weighed 230 grammes and was soft and very dark. The kidneys were pale, with a few cortical cysts. The pancreas, suprarenals, prostate and thyreoid were normal. Superficial yellowish-white necrotic ulcers were present on the soft palate and fauces. The intestines were normal, except for the anal fissure. There was no evidence of hæmorrhages beyond one small petechial patch on the buccal mucosa. Perl's Prussian blue test for iron was applied to five organs with the following reactions: liver and spleen, very strongly positive; kidney and pancreas, strongly positive; heart muscle, weakly positive.

Table I.

Date.	Time.	Erythrocytes in Millions.	Hæmoglobin Value (Sahli).	Colour Index.	Leucocytes per Cubic Millimetre.	Differential Leucocyte Count.			Reticulocytes Percentage.	Treatment.
						Granulocytes.		Lymphocytes.		
						Mature.	Immature.			
May, 1935.										
2		1·34	36	1·3	1,000	14	—	86	—	
9		0·99	27	1·4	—	—	—	—	<0·5	Liver.
11		1·09	22	1·0	—	—	—	—	1·0	Liver (16 cubic centimetres).
12		0·90	22	1·2	—	—	—	—	—	
13		0·92	20	1·1	—	—	—	—	1·0	
16		0·98	21	1·1	3,000	—	—	—	<1·0	
18		1·05	22	1·0	—	8	2	90	—	
20		0·96	23	1·2	2,900	—	—	—	—	Marmite.
21		0·75	17	1·1	2,300	—	—	—	<0·5	Transfusion (10 ounces).
23		0·72	19	1·3	1,100	—	—	—	—	
24	1 p.m.	—	19	—	1,000	—	—	—	0·6	Sodium nucleinate (one cubic centimetre).
	5 p.m.	—	—	—	2,000	10	22	68	—	Transfusion (10 ounces).
	9 p.m.	—	—	—	2,400	8	36	44	—	
25	9 a.m.	1·10	24	1·1	1,400	8	36	44	—	Leucocyte cream (one cubic centimetre).
	5 p.m.	1·10	22	1·1	2,200	—	—	—	<0·5	Liver (20 cubic centimetres).
26	11 a.m.	0·94	22	1·2	1,700	6	26	68	<0·5	Leucocyte cream (10 cubic centimetres).
	11 p.m.	—	—	—	2,500	—	—	—	—	
27	10 a.m.	0·99	22	1·1	1,500	8	28	64	<0·5	Leucocyte cream (10 cubic centimetres).
	7 p.m.	0·99	22	1·1	2,800	—	—	—	—	Sodium nucleinate (two cubic centimetres).
28	11 a.m.	1·03	21	1·0	4,200	—	—	—	—	Sodium nucleinate (two cubic centimetres).
	4 p.m.	—	—	—	7,300	—	—	—	—	

On microscopic examination the red bone marrow was comparatively cellular, with decrease of fat spaces. The cells were mainly reticular and endothelial, with many early precursors, myeloblasts and megaloblasts. Very few myelocytes were seen and few normoblasts—a picture of dyshæmopoiesis. The spleen was pigmented but not fibrotic, and contained foci of small reticular cells. The section of the liver showed increase of blood pigment. The kidneys showed no significant change. The cells of the pancreas were vacuolated and disintegrated.

Comment.

This case of macrocytic anæmia is considered worth reporting because it was extremely difficult to make an exact diagnosis. Recent progress in hæmatology has resulted in a fairly clear definition of most of the known hyperchromic or macrocytic anæmias, but this case presents an unusual combination of clinical features which does not conform to any one type.

On his admission to hospital the patient was thought to be suffering from pernicious anæmia in severe relapse. In fact, although the blood picture was slightly atypical, we were sufficiently confident of the diagnosis to delay the performance of a test meal pending the clinical improvement which was expected in response to liver therapy. The extract used was a well-recognized English preparation and the same batch was employed in the treatment of other patients with satisfactory results. The dosage should have been adequate, especially as it was complemented with liver given by mouth until late in the course of the illness. The reticulocytes were stained by a method similar to that used by Gardner and Wood,[1] and 1,000 to 1,500 red cells were counted on each occasion.

However, there was no reticulocyte response to liver, so, in view of the severe granulopenia, a diagnosis of aplastic anæmia was entertained. This, too, was discounted later by the outpouring of immature white cells which (possibly *post hoc* rather than *propter hoc*) followed the injections of sodium nucleinate and of "leucocyte cream". Additional evidence against aplastic anæmia was subsequently provided by the hæmosiderosis and appearance of the bone marrow *post mortem* and by the absence of hæmorrhages. A hæmolytic basis was suggested by the persistent pyrexia, hyperbilirubinæmia and hæmosiderosis, but these signs are not necessarily due to blood destruction, and there was neither the reticulocytosis nor the response to blood transfusion which is characteristic of Lederer's hæmolytic anæmia. Other marrow diseases, such as leucopenic myelosis (aleuchæmic myeloid leuchæmia) and chronic hypoplastic anæmia, were considered but readily rejected on the clinical and *post mortem* findings.

Ten years ago such a case would probably have been labelled "pernicious anæmia in an aplastic phase", but in the light of our present understanding of the ætiology of this disease it is difficult to see how there could have been a complete absence of response to liver in this case when the hæmoglobin before treatment was no lower than 27% (Sahli). There may have been an inability to utilize the maturation factor necessary for the development of the primitive red cell to the stage in which it is allowed to enter the blood stream. The case certainly bears a close resemblance to two of those described as "achræsthic anæmia" by Wilkinson and Israëls,[3] and to a similar case reported earlier by de Crespigny.[4] But it differs from these in having a much shorter course, more definite evidence of hæmolysis and no reticulocytosis at any stage.

Thus it seems almost impossible to explain all the features satisfactorily with a single diagnosis. Minot[5] referred to another instance in which the blood stream was practically devoid of embryonic red cells, although the marrow was hyperplastic; but in our case this apparent inconsistency was probably due to a fallacious presumption. Macroscopically the red marrow was definitely increased, which one is tempted to assume was due to erythroblastic hyperplasia. However, macroscopic appearances are often deceptive, and marrow smears are very difficult to interpret correctly, so it is more probable that there was an actual aplasia, or at least a hypoplasia of the erythroblastic marrow, and that the increase of hæmopoietic tissue was due to a delayed myeloid reaction alone. These changes could reasonably be attributed to some slowly acting toxic agent, probably a chemical poison, depressing the marrow and affecting the erythrocytes most severely, the granulocytes less so, and the platelets hardly at all. The increased hæmolysis which was almost certainly present might have been caused by the same agent.

Acknowledgement.

We are indebted to Dr. J. Gordon Hislop, the honorary physician in charge of this patient, for permission to report this history.

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(1) Hilda J. Gardner and Ian J. Wood: "Massive Dosage of Intramuscular Liver Extract in the Treatment of Pernicious Anæmia", THE MEDICAL JOURNAL OF AUSTRALIA, May 4, 1935, page 543.

(2) Max M. Strumia: "The Effect of Leucocytic Cream Injections in the Treatment of Neutropenias", *The American Journal of the Medical Sciences*, Volume CLXXXVII, April, 1934, page 527.

(3) John F. Wilkinson and M. C. G. Israëls: "Achræsthic Anæmia", *The British Medical Journal*, Volume I, 1935, pages 139 and 194.

(4) C. T. C. de Crespigny: "Observations on Cases of Rarer Forms of Anæmia", THE MEDICAL JOURNAL OF AUSTRALIA, September 30, 1933, page 439.

(5) George R. Minot: "Diminished Blood Platelets and Marrow Insufficiency", *Archives of Internal Medicine*, Volume XIX, 1917, page 1062.

Reviews.

OBSTETRICS AND GYNÆCOLOGY.

A COMBINED text-book of obstetrics and gynæcology has been produced by Munro Kerr and others.[1] It is written for the use of the student, and the style is simple and easily followed. The authors have made a point of answering the question "why?" in their descriptions of diseases, and this should be of great assistance to the student in memorizing facts.

In the chapter on anatomy, and indeed throughout the book, especially in pathology, more illustrations would be of benefit. The traditional English conservatism and distrust of new ideas until fully proven dominate the work; and some theories and methods that have been abandoned by the majority of obstetricians and gynæcologists are described and advocated. However, it must be remembered that the book was printed in 1933, and many theories of that time are now accepted proven facts.

The anatomy and physiology are elementary, and in the description of the pelvic floor the triangular ligament (a most important factor) is barely mentioned and is not described.

The obstetric section is quite well done, though again the student is advised to keep to the old well-beaten paths. Certainly if the young medical practitioner followed the precepts laid down in this book he would never go far wrong, though modern patients would probably demand more up-to-date methods of anæsthesia in midwifery.

The chapters on pathological labour and the puerperium are quite good, and obstetrical operations and their indications are excellently described.

The gynæcological section is not quite up to the standard of the obstetric section. Some chapters are excellent. The pathology, however, is not very modern. We were pleased to see that tubal insufflation is given a prominent place both in diagnosis and therapeusis, but that strict

[1] "Combined Text-Book of Obstetrics and Gynæcology: For Students and Medical Practitioners", by J. M. Munro Kerr, M.D., F.R.F.P. and S., F.C.O.G., J. Haig Ferguson, M.D., LL.D., F.R.C.S., F.R.C.P., F.C.O.G., F.R.S., James Young D.S.O., M.D., F.R.C.S., F.C.O.G., James Hendry, M.B.E., M.A., B.Sc., M.B., F.R.F.P. and S., F.C.O.G., with contributions from Charles McNeil, M.A., M.D., J. Duncan White, M.B., Ch.B., D.M.R.E.; Second Edition, revised, rewritten and enlarged; 1933. Edinburgh: E. and S. Livingstone; Sydney: Virtue Book Company. Royal 8vo, pp. 1112, with 497 illustrations and additional X ray plates. Price: £2 10s.

asepsis is insisted on and that the procedure is treated as a major operation.

Very many little points could be picked out in the book with which we are not quite in accord, but we must agree that the methods advised are at any rate safe, with the exception perhaps of the use of the Gilliam operation in retroversion. In our opinion this operation, if mentioned at all, should be wholly and completely condemned. The authors do make some slight reference to its risks. Certain modifications of the Gilliam operation are good and are not given enough prominence in the book. Indeed all gynæcological operations are very briefly described; this, however, is perhaps unavoidable in a text-book of this nature.

We also think the routine pre-operative preparation in operation cases is a little drastic. Surely a large dose of castor oil and two enemata are unnecessary in the majority of instances.

The authors do not seem to recognize that squamous carcinoma can arise from the columnar epithelium of the cervix by metaplasia, though they do state that "medullary" carcinoma arising from columnar cells can closely resemble squamous carcinoma. However, the metaplasia of columnar epithelium to squamous carcinoma is recognized in cancer of the uterine body. It would appear that "chronic glandular endometritis" is used as a synonym for *metropathia hæmorrhagica*, and the two terms are used in different places in a somewhat confusing manner.

On the whole the book is a safe, sound, conservative description of the practice of obstetrics and gynæcology and should be of great help to the student.

BALNEOLOGY AND CLIMATE.

THE mysterious rituals of hydrotherapy and spa procedures are exposed by Dr. Matthew B. Ray in his book, "Hydrotherapy and Climatotherapy".[1] Two-thirds of its contents are devoted to the principles and practice of balneology and the remaining third to the value of certain climates in disease. The author has apparently intended to write for the curious layman who has had perforce to interest himself in these forms of therapy, since a considerable amount of elementary physiology has been introduced and the rationale of physical medicine has been reduced to simple terms. Dr. Ray begins by a description of the skin and its functions. He then defines the value of baths in general and proceeds to give brief descriptions of special baths, their artificial preparation and objects. This section is adequately condensed and would alone render the book of value to the general practitioner. Types of mineral water, methods of analysis, constitution and sources, next receive attention. The various European spas are subsequently described, but in relation to their medicinal indications rather than to their geographic situations. Thus successive chapters are concerned with metabolic and gastric disorders, circulatory disorders, respiratory disorders, skin diseases *et cetera*. The section on climatotherapy is also concerned with the seasons and local meteorological and physical characters of health resorts in the British Isles, Europe and North Africa, with special reference to the types of conditions best treated at these places.

At first such information would seem to have little interest for Australian practitioners, unless they were asked by patients intending to travel to suggest the best itinerary for their particular case. Nevertheless the principles which govern the selection of a climate for a particular patient are equally applicable anywhere, and Dr. Ray has rendered his contribution of wider value by including them. Thus there are descriptive sections headed "The High Altitude Treatment of Pulmonary Tuberculosis" and "The Value of a Sea Voyage". In Australia we are very fortunate in the variety of climate available. It is not necessary to visit Cairo to obtain the equivalent of Egyptian sunshine, and one has merely to journey a few hundreds of miles to reach sufficiently elevated mountain conditions. In view of this lavishness of Nature, surely we should make a closer study of our local climate and its virtues and, with Dr. Ray's experience to guide us, to prepare in much the same way corresponding facts about Australia. Such information is badly needed in medical circles and travel agencies abroad, who are constantly being asked whether this or that part of Australia would be suitable for a particular patient. The British Health Resorts Handbook has an overseas section. The paucity of our knowledge of our own mineral springs and climatic resources has resulted in a deplorably small section dealing with Australia.

The book is unillustrated, but very readable. It would be a worthy investment by the general practitioner, even if solely to obtain the formulæ and directions for preparing baths, packs and douches. A short but laudatory foreword by Lord Horder points out that in this work "some spas are divested of their erstwhile extravagant claims and their benefits reduced to their practical values". It is the practical value of Dr. Ray's book which makes the deepest impression.

[1] "Hydrotherapy and Climatotherapy", by Matthew B. Ray, D.S.O., M.D., M.R.C.P., with a foreword by The Right Honourable the Lord Horder, K.C.V.O., M.D., F.R.C.P.; 1936. London: Edward Arnold and Company. Demy 8vo, pp. 319. Price: 12s. 6d. net.

ELEMENTARY PRACTICAL CHEMISTRY.

THE 1936 edition of "Salts and their Reactions: A Class-Book of Practical Chemistry", by Dobbin and Mackenzie, is a worthy addition to the series emanating from the University of Edinburgh.[1] The first production (1889) was based on notes originated by Professor A. Crum Brown for the guidance of his students in the laboratory. Between 1904 and 1913 Professor Hugh Marshall and Dr. Leonard Dobbin further elaborated the work, and subsequently, after the death of Professor Marshall, Dr. Dobbin and Dr. John E. Mackenzie collaborated in expanding it further and bringing it up to date.

The title of the book does not at first glance suggest the full scope of its contents, but the student who selects it as a guide to his studies in elementary practical chemistry will find all that he requires within its pages.

The first section contains, condensed in forty-seven pages, an exceptionally fine presentation of the fundamental properties of salt solutions. It deals with the ionization hypothesis and its many applications so clearly and succinctly and contains so much of importance to the chemical student that this section alone will render the book worth while to him. It is quite up to date and draws attention to the fact that the chlorides of sodium and barium are 15% less soluble in the recently discovered "heavy" water than in "ordinary" water. We must, however, be permitted a smile at the following rather obvious statement: "The abundance and cheapness of 'ordinary' water as compared with 'heavy' water make it improbable that its place in the laboratory will be taken by 'heavy' water except in very special experiments." We are reminded that Edinburgh produced the book.

The further sections, "General Methods of Preparing Salts", "Experiments Illustrating the Formation and Decomposition of Salts, together with some Simple Quantitative Measurements", "Reactions of Salts" (qualitative tests for basic and acidic radicles and scheme for qualitative analysis), "Dry-Way Reactions", "Volumetric Analysis", and "Experiments with some Common Organic Substances", are all excellently presented.

In an elementary practical chemistry book one looks above all else for that clarity and definiteness of expression which the nature of the subject demands, and one finds it in highly satisfactory degree in "Salts and their Reactions". The book can be confidently recommended to teachers and students.

[1] "Salts and their Reactions: A Class-Book of Practical Chemistry", by L. Dobbin, Ph.D., and J. E. Mackenzie, D.Sc.; Sixth Edition; 1936. Edinburgh: E. and S. Livingstone. Demy 8vo, pp. 255. Price: 6s. net.

The Medical Journal of Australia

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HOUSE SURGEONS AND SURGERY.

As time goes on the work of the surgeon is becoming more and more specialized. Surgical procedures in some parts of the body are becoming so complicated and the successful issue is so bound up with experience and judgement, that only those specially trained in certain tasks are qualified to perform them. In his president's address to the Royal Australasian College of Surgeons last March, Dr. R. B. Wade discussed the future of general surgery and expressed the view that in time to come the position of the general surgeon would be profoundly modified. How this modification will come about and in what way it may be controlled for the advancement of the art of surgery need not concern us at the moment. There is no doubt that Dr. Wade was right in his prophecy, but there are complicating factors that must be considered, especially in a country like Australia, with its long distances and its scattered population. A certain amount of surgery will always have to be undertaken by the general practitioner, and the average graduate should be so equipped that the opening of an abdomen (when he is called upon to relieve an urgent pathological condition) will not be a novelty to him. If he has been diligent in study and if he has been allowed to perform certain surgical operations under supervision, he will in ordinary circumstances be able to give a good account of himself. At the present moment we are not concerned with the ultimate range of the general practitioner's surgical efforts, a range that can be determined only by his own conscience. The training of house surgeons in the performance of ordinary surgical operations is made all the more important because of the unavoidable operations that will confront them in general practice. Of this Dr. Wade writes: "Should emergency operations be performed by the more junior surgeons? Are they not the cases in which mature surgical judgement is required?" Dr. Wade, in using these words, was speaking of hospital practice; they apply equally well to private practice.

The President of the Royal Australasian College of Surgeons recognizes the needs of the house surgeon who is to go into general practice. He states: "The house surgeon should be taught just that amount of simple practical surgery which needs no special post-graduate training, but which his Bachelor of Surgery degree entitles him to know, and which is common to everyday general practice." He points out that at the Royal Melbourne Hospital the work of the surgical associate (the graduate who is in training for the work of a general surgeon) does not interfere with that done by the house surgeon, who receives an adequate training in the surgical technique of conditions such as acute appendicitis and inguinal hernia. During the first month of each house surgeon's appointment he is not allowed to operate, and operations of the type just named are done by the associate. For the remaining months of each house surgeon's appointment he is allowed to perform operations of this kind under supervision. During 1935 the house surgeons at the Royal Melbourne Hospital performed over 360 operations of this type, the minimum number done by any one of the eighteen house

surgeons being fifteen. From information that can be gathered it appears that there are few teaching hospitals in Australia in which this procedure is adopted as a routine measure. The general conclusion may be stated that a teaching hospital is failing in its duty if it makes no effort to equip for the surgical emergencies of general practice house surgeons who by their keenness, aptitude and general bearing show themselves worthy.

The attitude of the house surgeon must be considered. He must remember that he is a pupil and is being taught. It is not for the bumptious and arrogant fledgling, when he is bidden to enlarge an incision or to handle tissues in a certain fashion, to reply that he prefers to do what is necessary in his own way. At least in the early days of his surgical career Jack is not as good as his master. Apart from any other consideration he should remember that the master, in teaching him, takes full responsibility for the end result of the procedure.

A final word for the honorary surgeons—they might well remember that there was a day when they themselves had to learn the rudiments of their craft, that their way was made difficult often enough because of the dog-in-the-manger attitude of their seniors, and that their honorary officership demands not only that they should treat the patient, but that they should be teachers of their resident medical officers as well as of their undergraduate students.

Current Comment.

EXTRACT OF SUPRARENAL CORTEX IN THE TREATMENT OF BURNS.

W. C. Wilson and two co-workers, G. D. Rowley and N. A. Gray,[1] have recently recorded their experiences in burns accompanied by acute toxæmia. In these columns during the past few weeks comment has been made on the fact that the skin of a burnt patient may suffer harm through the too enthusiastic use of tannic acid, and various journals throughout the medical world have expressed like views. In the present instance we presume to comment upon a method which, harmless in itself, may produce a favourable influence upon the worst feature of severe burns—the toxæmia which they engender.

At the outset we have to admit that the present report of Wilson and his colleagues is based upon the observation of three cases only, an amount of clinical material admittedly small. But we have to remember that the three persons under discussion were so situated as to render easy a comparison between their clinical state and that of other patients treated by other methods. Wilson has for some years past busied himself with an investigation into the causes of death following burns. His results, it seems, are to be published at an early date, and his observations will be no less welcome to us than, say, those of Cole upon the treatment of tetanus.

The treatment of the acute toxæmia of burns, though now less frequently necessary since the inception of the tannic acid treatment, must still be used to ward off death after extensive burns, especially in young children, who quickly develop unmistakable and fulminating signs. Strangely, no characteristic change in the blood picture heralds the onset of the toxæmia. But there are important clinical signs—those of a rapidly failing circulation. The acute signs of this nature, especially in young children, are unmistakable of their kind, and later we shall detail them. It seems certain that acute toxæmia results from the activity of circulating poisonous substances, the action of which is as yet a mystery, which are the derivatives of the autolysis of the injured tissues in the burned area. An increased concentration of the patient's blood, certain changes in the blood chemistry, and the early onset of bacterial infection are inconsistent accompaniments, but not necessarily essential causes of the toxæmic condition.

We must stress that our authors, in treating the seriously burned patients under discussion, have not departed from the accepted lines of treatment; they have attempted the immediate coagulation of burnt areas by the use of strong solutions of tannic acid; they have endeavoured to maintain the blood pressure at a normal level by the intravenous infusions of normal saline solution, gum and dextrose solution and of alkali. But in their experience this medication has, up to the present, proved ineffective in cases in which the toxæmia was severe and fulminating. Intravenous injection of the kind, they say, has proved of immediate benefit, but the improvement is of a few hours in length.

In the adult the manifestations of severe burning and the acute toxæmia following it are less dramatic than in children. The adult vomits and his temperature and pulse rate rise. If acute toxæmia follows the burns, there are quick mental changes—manifest anxieties, delusions, dull apathy and stupor. Death within one hundred hours generally follows these evidences of an acute toxæmia; but some adults survive their perilous first week or so to fall a ready prey to the added load of bacterial infection and the liver damage which appears during the second and third weeks.

[1] *The Lancet*, June 20, 1936.

Wilson and his colleagues, as we have already stated, quote the histories of three severely burned patients—a male child a little over three years old, a girl of five and a man of forty-nine. In each instance the burnt areas were cleansed under anæsthesia and the affected parts were covered with gentian violet (1%). Tannic acid, by various methods of application, had been applied, and the toxæmia had been predicted as the forerunner of a fatal result. In the case of the infant, "Eucortone", an extract of the suprarenal cortex, was administered in an initial dose of 0·5 cubic centimetre, followed by doses of 1·0 cubic centimetre at four-hourly intervals. Within half an hour of the first injection of 1·0 cubic centimetre the distressed and stuporose condition had been replaced by a state of quietude, the pulse rate had fallen from 200 to 120 per minute; the lips and ears, previously grey, had assumed a rosy tinge. Throughout the period of observation no change was discovered in blood chemistry or blood concentration. No organisms were discovered in blood specimens examined up to 72 hours. In the case of this infant the leucocyte count of 20,000 at the time of admission to hospital sank to 6,000 in one hundred and four hours.

In the second case under consideration, that of a child of five, the patient had suffered destruction by burning of 35% of her body surface. Within thirty minutes her skin had been cleansed under an anæsthetic and was undergoing frequent spraying with tannic acid. But the evidences of toxæmia became more and more manifest. The vomiting became continuous, the patient became cyanosed, the systolic blood pressure sank to 60 and the diastolic pressure to 40 millimetres of mercury. Within twenty-four hours so low was the blood pressure that it could not be estimated. "Eucortone" was now injected in doses of 0·05 cubic centimetre at intervals of from forty to sixty hours and thereafter in 1·0 cubic centimetre doses from sixty to ninety hours. Throughout no intravenous medication of any sort was employed. Within an hour, we are assured, the signs of acute toxæmia were waning and had disappeared entirely by ninety hours. The rise of the blood pressure was sustained and steady. A rise of temperature, indicating a mild bronchitis and bacterial infection, was the only indication of the course of the evil results of the burn.

Again without entering into prolix details, we briefly consider the third case of Wilson and his colleagues. The patient was a man of forty-nine years, severely and deeply burnt by fire over 25% of his body surface. At six hours there was no shock; at nineteen hours a small degree of secondary shock appeared which had vanished at twenty-nine hours. Then the rectal temperature and pulse rate rose and vomiting started. Nine hours later he was cyanosed, babbling, vomiting and hiccupping. The blood pressure was normal, but the corpuscular content of the blood was raised. "Eucortone" was given, first every two hours, then every hour for eighty-seven hours. The vomiting gradually stopped, the pulse rate and temperature fell, the skin became pink again, and the blood gradually showed a normal dilution. Subsequent evidences of intoxication were controlled by the use of "Eucortone", and the end-result was recovery.

There is no doubt that in this work of Wilson and his colleagues, combined with that of Dale upon the action of histamine on cats deprived of the suprarenal glands, we have to face a vast and increasing amount of evidence appertaining to many forms of reaction following removal of the suprarenal glands. Up to the present it would appear that no observations have been collected as to the use of adrenaline in burns. We need more accurate observation in casualty wards by admitting surgeons, the routine injections of suprarenal extracts following the admission of the burnt patient, and the closer observation by the hæmatologist and biochemist of the patient himself.

SPONTANEOUS PNEUMOTHORAX SIMULATING AN ACUTE ABDOMINAL CONDITION.

Clinicians know that thoracic conditions can give rise to such abdominal pain, tenderness and rigidity of the abdominal muscles that diagnosis may become extraordinarily difficult. The best known example, of course, is pneumonia. In a recent short paper, F. Steigmann and H. A. Singer[1] point out that other conditions which may be confused with the "acute surgical abdomen" are acute fibrinous pleuritis, coronary thrombosis, acute fibrinous pericarditis and certain lesions of the spine and spinal nerves. They also state that spontaneous pneumothorax masquerading as an abdominal affection is not known to medical writers; in only one book, by Adams, is the possibility mentioned. The authors have found records of four cases and in their own practices have had experience of no less than three. They attribute their discovery of these three cases to their routine use of radiological examination in all questionable or atypical abdominal conditions. The onset of symptoms in all three cases was acute; abdominal pain, nausea and vomiting, rigidity and tenderness were present. One patient died and no autopsy was made; the other two recovered. Attention is likely to be focused on the abdomen when the patient gives a history of previous gastro-intestinal disturbance. In four of the cases reviewed by Steigmann and Singer such a history was present. When an almost complete collapse of the lung has occurred, diagnosis will not be very difficult. When the pneumothorax is not very extensive, diagnosis may not be easy, even if the clinician has in mind its possible presence. X ray examination, carefully made, will probably remove all doubt.

[1] *The American Journal of the Medical Sciences*, July, 1936.

XUM

Abstracts from Current Medical Literature.

THERAPEUTICS.

Ascorbic Acid.

J. SENDROY AND M. P. SCHULTZ (*The Journal of Clinical Investigation*, July, 1936) report investigations into the effect of ascorbic acid in preventing rheumatic fever or acute rheumatism. First, tests were carried out which showed that with a given intake of ascorbic acid, as in orange juice, excretion in the urine increased proportionately and continued for some days after ingestion ceased. Ascorbic acid was administered to patients with rheumatic fever and to normal subjects; the urine was tested and it was shown that no definite evidence of ascorbic acid deficiency occurred in the rheumatic patients. In a further experiment ascorbic acid and orange juice appeared to have no effect on the manifestations of rheumatic fever.

Peptic Ulcer.

C. A. FLOOD AND C. R. MULLINS (*American Journal of Digestive Diseases and Nutrition*, July, 1936) record the results of treating twelve patients with saline injections, and six with histidine, for persistent peptic ulcer. Five cubic centimetres of normal saline solution were injected daily for three weeks in one group, and five cubic centimetres of a 4% solution of histidine monohydrochloride in the other group. Eight of the twelve patients treated with saline solution had complete relief of pain during the treatment; five of these remained quite well for one to four months. Of those treated with histidine, four improved and became free of symptoms, except for complaints of "gas" at times; two became worse. Three of these patients remained well and three had recurrence of symptoms. The authors conclude that saline solution is as effective as histidine in the treatment of peptic ulcer, and suggest that improvement depends on a moral effect.

Sodium Chloride in Diphtheria.

HOLDING the view that in diphtheria there is a considerable deficiency in the secretion of the suprarenal cortex, Alex. MacLean (*The Lancet*, July 18, 1936) reports the results of an investigation to observe the effect of the administration of sodium chloride. His cases were grouped according to the degree of general intoxication and the severity and extent of the local lesion. After a close analysis of his figures he concludes that the administration of extra sodium chloride to patients with diphtheria is associated with an improvement as compared with the condition of patients not receiving extra sodium chloride. The author points out that it has long been recognized that suprarenal hæmorrhage is common in fatal diphtheria; cortical changes are almost invariably seen when there has been severe intoxication. The general symptoms in the acute stage of diphtheria are consistent with symptoms of suprarenal cortex deficiency. The biochemical changes noted, particularly those in the carbohydrate metabolism, are also consistent with deficiency in the secretion of the suprarenal cortex. The author has observed evidence of a deficiency of sodium and chlorine in the blood serum in the acute stage of diphtheria. The expense of cortical extracts prevents any adequate trial of these preparations in this disease.

Idiopathic Headache.

L. A. CRANDALL, G. M. ROBERTS AND L. D. SNORF (*American Journal of Digestive Diseases and Nutrition*, July, 1936) describe the results of treatment of 184 patients with classical migraine or other types of idiopathic headaches by chondroitin. Chondroitin sulphuric acid is a compound of glycuronic acid, galactosamine and sulphate groups. Doses of two to four grammes (32 to 64 grains) three times a day, before meals, were prescribed. During the administration of chondroitin migrainous headaches were less frequent and less severe as a rule, and other headaches were often similarly relieved; but when chondroitin was suspended the headaches soon returned in a majority of cases, though a few patients remained free from headache for three to six months.

Narcolepsy.

M. PRINZMETAL AND W. BLOOMBERG (*The Journal of the American Medical Association*, December 21, 1935) record the effect of treating narcolepsy and cataplexy with benzedrine, a preparation allied to ephedrin and epinephrin. Benzedrine β-phenylisopropylamine was more stimulating to the central nervous system than the other preparations. Nine patients who had typical attacks of narcolepsy were treated. These patients were prone to fall asleep regularly three to five times a day, and several had lost their work through this symptom. From 10 milligrammes once a day to 40 milligrammes three times a day were given. All the patients were relieved. The only untoward effect was that some of the patients could not sleep at night-time while taking the larger doses. A reduced dose was found satisfactory in these cases.

Prostigmin in Myasthenia Gravis.

CONSIDERABLE experience in the dosage and frequency of administration of prostigmin in *myasthenia gravis* has been acquired by L. P. E. Laurent and Mary B. Walker (*The Lancet*, June 27, 1936), who report their results and details of a follow-up of eight cases. When given by mouth, the drug was found to be effective only in much larger doses than those required for parenteral administration, 25 to 30 milligrammes giving a result comparable in intensity and duration with an injection of 0·5 milligramme. Inadequate supplies prevented further investigation of oral therapy. An analogous drug, "Substance 36" of Aeschlimann and Reinert's series, has been used orally in increasing doses with success. A dose of 10 milligrammes gave a demonstrable though weak result; gradually increasing doses up to 90 milligrammes were given without pain or unpleasant side action; even 120 milligrammes and 150 milligrammes have since been administered successfully. The best results are obtained with the larger doses, an improvement being noticed in forty-five minutes after administration; the maximum benefit is obtained in two hours and is maintained for six to eight hours, then gradually wearing off. The oral administration has much to commend it: it obviates the necessity for injections; it gives a more prolonged action than any method previously employed, and it would be possible to keep the patient at her best for twenty-four hours by giving two or three doses. The experiences with eight patients show that the drugs of this group can be taken for very long periods without producing any unpleasant symptoms or leading to any deterioration in the myasthenic state of the patient. Abdominal pain can be avoided by the simultaneous administration of atropine with the larger doses of prostigmin.

"Plasmoquin" and "Atebrin".

R. N. CHOPRA, J. C. GUPTA AND B. SEN (*The Indian Medical Gazette*, June, 1936) have carried out investigations into the relative value of "Atebrin" alone and "Atebrin" and "Plasmoquin" combined, in the treatment of malaria. They conclude that in benign tertian and quartan malaria the combination is no more effective than "Atebrin" alone, but in malignant tertian the two drugs combined cause a more rapid disappearance of parasites from the peripheral blood. In all forms of malaria the relapse rate, in the authors' series, was lower when the combination was employed. The combination was no more efficient than "Atebrin" alone in the reduction of the size of the spleen. There was less risk of toxicity when "Atebrin" was given alone.

NEUROLOGY AND PSYCHIATRY.

Insulin Treatment in Psychiatric Cases.

A. E. BENNETT AND E. V. SEMRAD (*The American Journal of Psychiatry*, May, 1936) report the results of an investigation into the value of insulin therapy as an adjuvant in the management of malnourished psychiatric patients. Twenty-five underweight

psychiatric patients (nine males and sixteen females) were given insulin, and their weight gain was compared with that of twenty-five patients (thirteen males and twelve females) on ordinary fattening diets, but who did not receive insulin medication. The ages varied from fifteen to seventy years. All patients were undernourished according to their normal weights. The group included both psychoneurotic and psychotic cases, the latter being in the majority. All patients had an intact carbohydrate metabolism. The patients were all ambulatory and participant in regular psychiatric ward activity. Insulin was usually given fifteen to twenty minutes before meals, two or three times a day. The initial dose was usually five units, increased to ten units and fifteen units if the patient's weight gain was not satisfactory. The 25 patients receiving insulin averaged a gain of 0·5 kilogram (1·09 pounds) per week. In the control group, without insulin, under the same conditions, an average gain of 0·31 kilogram (0·673 pound) per week was made. Thus the patients treated with insulin gained 0·19 kilogram (0·42 pound) per week more than the patients treated under the same conditions without insulin. The greatest single gain obtained was one of 16·0 kilograms (35 pounds) in an involutional depressive patient. Sixteen patients in the insulin group gained more than 5·0 kilograms (10 pounds). Only six patients in the control group gained more than 5·0 kilograms (10 pounds). The most significant weight gains were seen in the affective reaction types. There was an average gain in the manic-depressive group of about 6·0 kilograms (14 pounds). The desirability of a method that will insure such gain in weight in the depressive patient, who often presents the most difficult feeding problem, is stressed by the authors.

Malaria Therapy.

G. M. Davidson (*The Journal of Nervous and Mental Disease*, July, 1936) suggests that the mechanism of malaria therapy in general paralysis is one of reticulo-endothelial reaction to the malaria parasite. Pathological studies demonstrate changes in the reticulo-endothelium of the nervous system following malaria therapy, among which are a reduction in the neuroglial reaction and a macrophagic response, particularly in the leptomeninges. The reticulo-endothelial apparatus in various other organs, liver, spleen *et cetera*, is stimulated. For the estimation of the function of the reticulo-endothelial system the author has employed the Congo-red and colorimetric method. An index of 50% to 70% was considered normal. In two untreated cases of general paralysis the Congo-red index was 90% and 95% respectively; in two cases in which malaria therapy was employed with good results, it was 75%. The test thus demonstrates a definite hypofunction of the reticulo-endothelium in general paralysis and improvement following malaria therapy, which falls in line with the pathological findings. Finally, the author suggests that these conclusions as to the rôle of the reticulo-endothelium may throw light on the question of the ætiology of general paralysis. Various predisposing causes, such as alcoholism, trauma *et cetera*, may act by producing reticulo-endothelial dystonia, and the individual constellation of the reticulo-endothelial system, whether congenital or acquired, may be the underlying basis of the small percentage of syphilitics who develop general paralysis.

In order to elucidate the question of the participation of a humoral defence mechanism in the curative effect of malaria fever, experiments were undertaken by A. Beek (*The Journal of Mental Science*, May, 1936), comprising: (i) examinations of the behaviour *in vitro* (immobilization, eventual agglutination) of *Spirochæta pallida* in the presence of serum and cerebro-spinal fluid of malaria-treated general paralytics; (ii) examinations *in vivo* on the existence of a bactericidal substance (spirochætocidin); and (iii) examinations of a phagocytosis-enhancing substance (spirochætotropin) in the serum and cerebro-spinal fluid of these patients. The sera were also examined for the antibodies recognizable by the adhesion test (Brown and Davis). The samples of blood and cerebro-spinal fluid were derived from patients who had undergone treatment with malaria from two to six months previously. As antibodies would be most likely to be present in improved cases, patients were chosen for the experiments who had already shown some clinical evidence of improvement. No evidence of the existence of antibodies against *Spirochæta pallida* in the serum and cerebro-spinal fluid of malaria-treated paralytics was obtained. The author concludes that the mechanism leading to the improvement of general paralysis is not of humoral, but of cellular nature.

Hæmatoporphyrin Treatment of Melancholia.

J. Huehnerfeld (*The American Journal of Psychiatry*, May, 1936) summarizes the known facts relating to the hæmatoporphyrin treatment of melancholia and endogenous depressive disorders. In the dosage employed by the author, hæmatoporphyrin exhibits a photodynamic, stimulative-tonic, and vegetative-regulatory action. In the treatment of melancholic and endogenous depressive conditions the photodynamic effects of the dyestuff manifest themselves particularly in the cases of severe inhibition. Often a few days' treatment suffices to bring about a relaxation of the condition, inhibition regresses, and the movements, facial expression and vocal expressions become livelier. No particular source of light is employed along with the hæmatoporphyrin treatment. The stimulative tonic action is shown by striking improvement of the appetite, increasing body weight and, in attendant secondary anæmia, by an improvement in the blood picture. In many cases the skin changes are particularly striking. Soon after the institution of the treatment the skin becomes more tense and shows increased turgor. The vegetative-regulatory action is manifested by improved salivary secretion and regression of the frequently occurring gastro-intestinal disturbances and unpleasant symptoms, such as fullness of the abdomen and a sense of oppression in the chest. A favourable influence is also exerted on functional cutaneous disturbances, such as sweating, changes in sebaceous gland secretion, and conditions based on vasomotor disturbances. The increased calcium and decreased potassium values, which are often found in melancholic patients, return to the normal. The treatment is indicated chiefly in the depressive disorders of the manic-depressive domain. Exogenously conditioned depressions will respond to the treatment only if endogenous factors participate, though but slightly, in the disease picture. The treatment is not suitable for the depressive phases of schizophrenic psychoses. Among the patients who are influenced only slightly or not at all are to be found mostly those with predominant anxiety complexes and lively hallucinatory elements. The treatment is contraindicated only in the presence of severe acute febrile conditions or organic liver affections. Up to the present, 400 cases have been described. Among them are 327 consisting for the most part of severe forms of melancholia and endogenous depression. Of the 327 patients, 98 were distinctly improved, 162 cured or greatly improved, and only 67 entirely uninfluenced. The author concludes that it is permissible to ascribe to hæmatoporphyrin a specific influence on the melancholic and depressive conditions.

Familial Schilder's Disease.

A. Meyer and T. Tennant (*Brain*, Volume LIX, Part I, 1936) report in detail familial cases of Schilder's disease, occurring in two sons and the mother. The illness of the mother did not manifest itself before the onset of the disease in her children, and although a definite diagnosis of her condition is not yet made, she exhibits a type of disseminated sclerosis or an otherwise unknown type of progressive spinal disease. Gastro-intestinal troubles were observed in the two brothers long before the nervous affection was manifested. Another brother at present presents these gastro-intestinal symptoms and has also a slight ataxia. No histological changes were found in the viscera to account for the involvement of the central nervous system. The ætiology and pathogenesis of Schilder's disease are discussed at length and a theory of primary glial affection is advanced.

XUM

Medical Societies.

THE MELBOURNE PÆDIATRIC SOCIETY.

A meeting of the Melbourne Pædiatric Society was held at the Children's Hospital, Melbourne, on June 10, 1936. Parts of this report were published in the issues of October 10 and October 17, 1936.

The Results of Conservative Treatment of Tuberculosis of the Hip.

Dr. J. B. Colquhoun read a paper on the results of conservative treatment of tuberculosis of the hip. He said that in the time at his disposal he could allude but briefly to the treatment employed in the securing of results in this condition, the management of which was so difficult and so distressing to the inexperienced. He would, however, allude to the general outline of treatment, so that those present might appreciate what it entailed. After careful clinical, biochemical and radiological examination, diagnosis of tuberculosis of the hip was arrived at and the patient was prevented from bearing weight or doing anything strenuous. The first principle of treatment was rest, which had to be prolonged, continued and uninterrupted, and which had been advised over half a century ago by H. O. Thomas in his classical treatise on that subject. The second principle of treatment was a regimen of life out of doors all the year round, combined with systematic heliotherapy. The third principle was an adequate diet, and the fourth was education and an occupation of some kind. The rest was to be insisted upon until no further signs of the disease were observed; at the end of that stage of rest there began a period of ambulatory treatment which extended until all apparatus was abandoned and the patient took up a normal and active life once more. Dr. Colquhoun said that he would not refer in any detail to the life out of doors, or to the problems of education, occupation or dietary employed, but he would confine his remarks to a description of how the required rest for the diseased part was secured and the results he had obtained.

At Frankston, where most of the work had been done, each new patient was put into a plaster of Paris spica, extending from the lower ribs to the toes on the affected side. The hip was placed in the neutral position as regards abduction and adduction, with slight hip flexion and internal rotation; the spica was moulded with meticulous care over the iliac crests, the greater trochanter, the ischial tuberosity, the patellar and knee regions, and about the malleoli. Dr. Colquhoun said that with these casts any starting pains that had been present invariably disappeared. The cast was removed every six months and a radiograph was taken, after which the case was reviewed. That stage of treatment generally lasted for about two years. If the starting pains persisted while the limb was in plaster, then the plaster was removed and traction was applied in one of the many various ways; these pains did not last long and the patient was put back again into a plaster spica. Dr. Colquhoun said that the abscesses that formed during this stage were interfered with only if they threatened to break down, and that simple aspiration was all that was necessary; any sinuses that formed were treated surgically and had usually healed in several weeks' time. When the ambulatory stage was reached, the patient gradually started to bear weight by holding onto the bed in the ward. At that time the affected limb was encased in a long plaster spica with a walking iron of the Frankston pattern attached to it. Careful watch was kept on the pulse, temperature, weight and sedimentation rate, and if these were found to be satisfactory the patient was discharged from hospital. The sedimentation test was, in Dr. Colquhoun's opinion, one of the most satisfactory tests of activity in surgical tuberculosis. So much so, that if the rate of sedimentation increased he would proceed slowly with activity until the cause of the increase was discovered and the rate returned to normal limits. In the ambulatory splint the children could get round very well at home and the parents were apt to ask before many weeks whether the child could go to school. Many of the children did attend school, but some of them had played so hard that they had broken their walking iron or plaster. Lately Dr. Colquhoun had been employing the Thomas hip ambulatory splint, because of the many patients he had walking around in plaster, which they had broken too frequently. He did not consider that splint as good as the walking spica, for, no matter how high a patten had been used, naughty children had walked around bearing the full weight on the diseased joint. Besides, it could be too easily removed by a patient or his parent as soon as they were out of sight. Dr. Colquhoun was sorry if he had wearied his audience with unnecessary details, but he insisted that only by strict attention to the above routine had he been able to secure such uniform results in this condition. Dr. Colquhoun said that on visiting Carshalton, where many of the tuberculous patients in London were treated by Dr. Pugh, he had been informed that Dr. Pugh did not trust a tuberculous hip unless sinuses had developed and healed or the abscess had been aspirated. Dr. Colquhoun thought that Dr. Pugh said that he had not seen bony ankylosis occur in a tuberculous hip when this had not taken place. He said that in his own list of cases it had occurred several times, and he would demonstrate these cases later by lantern slides.

During a visit paid to the orthopædic section at Frankston Sir Henry Gauvain was asked how long, on the average, it took to heal tuberculosis of the hip at Alton, and he had replied that it took about one year. Dr. Colquhoun said that unfortunately neither his colleagues nor he could claim such spectacular results in the sunshine of Victoria. Tuberculosis of the bones and joints ran a course, as a rule, under ideal conditions, from the beginning of activity until it became quiescent, which lasted at least two years. After that time, in tuberculous disease of the hip, a further period of protection was required, lasting for about two years, before bony ankylosis resulted. That period of protection had to be greatly increased when the patient was without ideal surroundings and treatment. If the softened tissues of the quiescent hip were subjected to the stress and strain of weight-bearing without protection, flexion and adduction would inevitably result, even if the disease was not reactivated. Dr. Colquhoun said that he would like to show fifteen of the patients who had been undergoing the treatment at Frankston which he had just been describing, and the results he had obtained. He described their histories in detail and also included two who were not present. They may be summarized briefly:

1. L.P., a female, after two years and eight months in hospital, was discharged in November, 1932. During treatment she developed sinuses which healed within six months; the result at the end of treatment was solid bony ankylosis, she walked with a slight limp, but no protection, and had half an inch of shortening.

2. M.S., a female, after six years in hospital, was discharged in July, 1934, with ankylosis of the right hip and the spine. In 1931 the radiogram showed solid bony ankylosis of the hip, three years after the institution of treatment; she had not developed any sinuses. The response to the Mantoux test was positive and the blood sedimentation rate was seven on discharge, and there was a shortening of one inch.

3. J.V., a male, after six years in hospital, was discharged on April 17, 1934, after having tuberculosis of both hips. He did not develop any sinuses and both hips were ankylosed. The Mantoux test gave a positive reaction.

4. H.G., a male, was admitted to hospital with a tuberculous thoracic spine and right hip; he received nearly ten years of treatment and was discharged in January, 1936. The hip was ankylosed and quiescent long before the spine, and there was considerable shortening.

5. F.R., a male, had developed tuberculosis of the left hip at the age of eight months and then of the right elbow; he was in hospital for four and a half years and was discharged in 1934 with bony ankylosis in the hip and a shortening of three-quarters of an inch; no sinuses had developed.

6. N.J., a female, was treated first in a Thomas abduction splint and then in plaster for approximately four years. Sinuses developed, but the hip healed with a good range of movement; there was a shortening of two inches.

7. M.M., a female, developed sinuses and flexion deformity of 60°; subtrochanteric osteotomy was performed. There was solid ankylosis and she had one inch of shortening.

8. C.P., a female, was treated by extension and developed a definite flexion deformity and adduction. There were great destruction of the head and neck of the femur, and, after ten years, fibrous ankylosis. Subtrochanteric osteotomy was performed and there was a shortening of two and three-quarter inches.

Dr. Colquhoun said that all the patients he had already mentioned were walking around without any splint or protection. He then discussed a further series in the ambulatory stage of treatment, who had all been under his constant care since the onset of tuberculosis.

9. E.T., a female, was discharged from hospital after four years, in a walking spica. At the time of the meeting the patient was in a Thomas hip splint. The hip was ankylosed and no sinuses had developed, but there was one and a half inches of shortening.

10. N.W., a male, had been in hospital for three years with tuberculous disease of the right hip, and was discharged in a walking spica on December 2, 1934. The hip was ankylosed and no sinuses had developed; there was a shortening of two inches.

11. J.M., a female, with tuberculosis of the right hip, had been discharged from hospital after three years of treatment on December 2, 1934, in a walking spica. There were no sinuses and the patient had one inch of shortening. At the time of the meeting she had a Thomas hip splint and the hip was ankylosing.

12. J.Y., a male, was admitted to hospital with tuberculosis of the right hip; sinuses developed, and he was discharged in a walking spica after three years and six months, on March 25, 1935. The patient had been at Fairfield Hospital with diphtheria for nearly a year, and on his discharge from there ankylosis was developing.

13. M.K., a female, was admitted to hospital with very acute tuberculosis of the left hip; no sinuses developed, and she was discharged in a walking spica, with a shortening of one and a half inches, on December 2, 1934, after two years and ten months, and at that time ankylosis was developing.

14. V.K., a female patient, had tuberculosis of the right hip and had developed sinuses, and the hip had ankylosed. She was discharged on December 1, 1934, after two and a half years, with a shortening of two and a half inches, and was now in a Thomas splint.

15. M.B., a female, was admitted to hospital with tuberculosis of the right hip and had no sinuses; she was discharged, after being two and a half years in hospital, on February 3, 1935, and ankylosis was developing at that time. The patient was in a Thomas splint at the time of the meeting.

16. M.O'M., a female, was admitted to hospital with tuberculosis of the right hip; she remained in hospital for one year and ten months, the ambulatory period being six months in a walking plaster. She was very destructive on plaster casts, and was at the time of the meeting in a Thomas hip splint, walking with crutches and patten. No sinuses had developed and ankylosis was progressing.

17. J.K., a male, had been in hospital for three years with sinuses in and from the hip; he was, at the time of the meeting, walking in a Thomas splint, and ankylosis was proceeding.

Dr. Colquhoun said that he offered no apology for presenting the small series of cases to the meeting, for, he said, each patient had had a great deal of his personal attention and care, and he considered that on that account the results that were shown were of value. Dr. Colquhoun pointed out that in only one case out of the seventeen had healing taken place with movement. Healing with movement was so rare in tuberculosis of the hip that most observers distrusted the diagnosis. Dr. Colquhoun said that in that particular case the diagnosis could not possibly be false, because of the sinuses and the clinical course of the disease. He wondered if, in view of the excellent result obtained in that case, they should not strive to get healing with movement. Rollier had claimed that he got a higher percentage of cures with movement, but in view of the danger involved in securing that result, and the relatively few cases in which such movement was of value, Dr. Colquhoun felt that solid bony ankylosis was the result to be aimed at.

This series of cases showed ankylosis proceeding equally in those patients who had not developed sinuses as in those who had. While sinuses had developed in seven out of seventeen cases, or in 40%, they had also healed in every case except when the sinus had tracked into the rectum. Shortening had, of course, been definite and had averaged about two inches, but shortening was one of the most constant deformities in tuberculosis of the hip.

Dr. Colquhoun said that since he had arrived in Australia there had been no mortality among the patients with whom he had come in contact, and who had been treated conservatively. Multiple lesions had been common when immobilization was not adequate in the treatment of tuberculosis of the hip. In four of the seventeen cases (25%) other lesions had been present, but these had been seen only in patients who had not been properly immobilized in the beginning. Dr. Colquhoun said that dislocation of the hip had not occurred in any case that he had seen. He believed that in the treatment of tuberculosis of the hip in children the conservative method was the method of choice and that solid bony ankylosis should be aimed at and could be secured, even if sinuses did not form. Conservative treatment could produce a good result in a little more time and with much less mortality than operative treatment could, and there was less danger of secondary infection with consequent complications; also there was the possibility of securing a movable hip as an end result. In securing consolidation of a healed tuberculous hip with bony ankylosis, protected weight-bearing in a walking spica was the best method of hurrying this end result.

DR. J. G. WHITAKER described the subject as a fascinating one, and thanked Dr. Colquhoun for the presentation of it which he had given that night, showing the results he had obtained. It was only fair to say that Dr. Colquhoun would not like to go away from the meeting leaving the impression that the method he had put forward was the only way of carrying out conservative treatment of tuberculous disease of the hip. Dr. Whitaker was worried by the fact that these patients pursued such a tempestuous course; he often wondered whether tuberculous disease was necessarily and unavoidably a disease with a course of two to four years, with destruction of the joint and such a doubtful end result. Dr. Whitaker showed two skiagrams, the first of which was almost of normal appearance; the other, taken after ten months of conservative treatment at Frankston, under his supervision, demonstrated that, in spite of his closest attention, the little boy's hip had become disintegrated. At present, Dr. Whitaker had sixteen patients with this disease under his care at Frankston, and under his very eyes in practically every case dissolution of the hip joint with wandering acetabulum was taking place while the patient was lying in the plaster. It was not surprising that he was in doubt concerning the soundness of the method adopted or that he should wonder whether some other conservative method, such as one in which traction was incorporated, should be substituted. Another problem was the criteria that should be regarded as essential to a decision to interfere operatively; he had wondered whether operative interference should be undertaken before destruction of the joint occurred. From his reading and from his inquiries it would appear that no English-speaking orthopædist was able to decide the point. His colleague, Dr. Osborn, had taken the bull by the horns some time ago, and it would be interesting to hear from him an account of his experiences. Dr. Whitaker himself had had some encouraging results and some failures; but it was indisputable that some gruesome

results had followed conservative treatment. He displayed a skiagram of a little girl who had been six years in hospital at Frankston under treatment in what could be regarded as ideal conditions; her case had proved a severe one, with total destruction of the hip joint and associated disease of the spine, and at present she had five inches of shortening.

DR. ROBERT SOUTHBY asked Dr. Colquhoun whether he made a point of including an adequate amount of calcium and of vitamins *A* and *D* in the diet of the children undergoing the conservative treatment he had described. He also asked whether, in the series of children investigated by Dr. Colquhoun, there had been any instances of an underlying specific basis, and, if so, to what extent, and whether these children responded better if they were given antispecific treatment. He also asked Dr. Colquhoun how he could tell in such cases that the hip condition was tuberculous rather than syphilitic.

DR. W. KENT HUGHES said that he thought that similar results could be obtained with the use of more heliotherapy and less plaster, which excluded the sun's rays from the skin it covered. Dr. Kent Hughes was wedded to exposure of as much of the body as was possible; he had been experimenting with thorough heliotherapy at Warrandyte. Here certain tramway-men with tuberculosis had a sylvan retreat where they could walk about in a pair of boots and little else; they slept in hammocks without clothes, even through the winter, and they did very well. Dr. Kent Hughes referred to a visit he had paid to Pinna Hospital with Sir Robert Jones, where he saw a number of patients with spinal tuberculosis without splints, prone on sleeping boards, unclothed and bronzed, even in the climate of England. He was of opinion that if the hospital were on a hill instead of at the beach, as was the case at Frankston, better results would be obtained from heliotherapy.

DR. DOUGLAS GALBRAITH said that during the past two years at Frankston he had been interested to watch the progress of the children undergoing treatment for tuberculous disease of the hip joint. Like Dr. Whitaker, he had been struck forcibly by the fact that the disease seemed to go on progressively under what one could regard reasonably as most favourable possible conditions. He had been surprised to see how little was the toxæmia that accompanied the progressive bone destruction. He had carried out many estimations of the rate of blood sedimentation on these patients and could state that it remained normal during the period of immobilization. It had proved a valuable guide when the patient started to get up and move about, and was regarded at Frankston as a useful and reliable control of the amount of freedom of movement that could be allowed. One aspect of the toxæmia was of interest: in these cases it was so different from the early evidence of the toxæmia which could be demonstrated in pulmonary tuberculosis; in tuberculosis of the hip the toxæmia seemed to run in cycles; the patients did not get a daily dose of tuberculin from the lesion; it seemed logical to use tuberculin therapeutically. E. D. Telford, of Manchester University, had reported similar results and stated that he had recently seen three patients whom he had treated in childhood; one was a motor bus conductor, another was a driver, and the third was a brewer's dray man; all had ankylosed hips, but each was able to do his job and had kept remarkably well.

DR. C. H. OSBORN said that although the series was not large Dr. Colquhoun had at least followed the progress of the patients closely for years. Dr. Osborn thought that a surgeon should feel reluctant to express an opinion on such a subject before he was on the wrong side of sixty years and had seen patients reach adult life and was in a position to be sure of the prognosis. Failing this qualification, he could only talk from evidence in books and correlate his experience with that of other people. With his own small series that he had watched over a very few years, he had decided to set a line of treatment for himself and see what happened. He felt that the case that Dr. Colquhoun had made out was subject to some criticism. In the first place it was extraordinarily difficult to know clinically when a hip joint was ankylosed (and by that he meant true bony ankylosis); skiagraphic evidence had to be sought. Certain of the skiagrams that Dr. Colquhoun had shown presented very characteristic and unmistakable appearances of ankylosis, but some of them were by no means so conclusive; frankly, in one of them he could not see the evidence. These children might proceed to the stage of indisputable bony ankylosis, but, on the other hand, breaking down might occur perhaps after an interval of years. Another point open to criticism was that only two of the children seemed to have bony ankylosis with a small amount of shortening, say under two inches; all the others had great shortening, amounting to three, four or even six inches, with an immense amount of destruction. In the latter cases the condition had been rather troublesome and the children would be seriously handicapped; even then Dr. Osborn was not sure that some of them had firm bony ankylosis; they might yet have further trouble ahead of them. In a wave of enthusiasm, impelled by the desire to improve the position, Dr. Osborn had commenced some time ago to operate in the early stages. It might be recalled that some six or seven years ago it had been the vogue in the surgical journals to describe operative technique for bone grafting in all sorts of ingenious ways to get fixation of the hip joint without disturbance of the diseased area. Dr. Osborn had selected for trial the technique of extraarticular arthrodesis of the hip joint culled from the American medical Press, and had started out with a small series of four or five patients. For a while he had been very happy about them; however, all the grafts subsequently broke; they had appeared to do well for three or four years, but the patients still had unfixed joints. He had allowed the foci to become quiescent and then performed the operation; subsequently he had subjected the patients to prolonged immobilization. In one case the patient had broken his graft at the age of sixteen years. In face of these facts Dr. Osborn had given up extraarticular arthrodesis and had started to use intraarticular methods of arthrodesing the joint, after getting assurances from Girdleston, of Oxford, and other sound conservative judges, such as Goindry, of the Mayo Clinic. He placed a bone key across the joint in a slot which he formed from the ilium into the head and neck of the femur. The operation could be done quickly and with little shock to the patient. So far he had done about five of these operations, but it was far too early to know what would be the outcome, though he could say that one had failed. It had struck him that ideals for treatment required good, sound bony ankylosis; those patients who had not got it were under a cloud up to the fourth decade. It was not wise to operate in the early stages, but, during that stage, Dr. Osborn did not think that it was ideal to use plaster immobilization alone; the plaster did not control sufficiently the upward trend of the head and the wandering of the acetabulum; in the successful cases gross shortening was present. Pugh's continual extension in the active stage was being tried again, but Dr. Osborn was not sure that it would be successful. Moderately good immobilization could be obtained in a trough of plaster with extension, and surely less shortening and deformity would result. When the activity had settled down, Dr. Osborn felt that it was a fair thing to operate with the object of stabilizing the joint; at least longer legs and more certain bony ankylosis would result. The operation should be followed by a period of immobilization for upwards of twelve months; if the skiagram then showed sound bony ankylosis, the patient could be allowed freedom to do anything, and so the period of inactivity would be shortened. Patients should be walking at the end of two years without appliances and without risk. Of course, Dr. Osborn wished it to be understood distinctly that this opinion was subject to retraction later on, but the uncertainty of more conservative treatment had led him to be an advocate for more active treatment.

In reply to the discussion, Dr. Colquhoun said that he was not anxious to pick up the gauntlet that night; in no case that he had treated himself had dissolution occurred. He had had much experience in the interpretation of skiagrams in this condition, and he felt able

to tell by clinical examination and the radiographic appearances when it was certain that bony ankylosis was present. He was willing to admit that some of the skiagrams gave more definite evidence than others, but he had not claimed that this desirable result had been reached without feeling morally certain that such was the case. In this disease they had to deal with two factors, the organism and the soil, and in the small series under discussion he might have been fortunate. It was no good saying that traction had failed if this opinion was based on the results obtained at Brighton Convalescent Cottage before the Frankston Orthopædic Branch was open. Between the infrequent visits of the honorary medical officers to Brighton the children took all sorts of liberties, and he had satisfied himself that the traction was not maintained and that the hip joints were not at rest. With reference to the remarks of Dr. Kent Hughes about plaster and heliotherapy, all that Dr. Colquhoun could say was that he was certain that the patients on Thomas splints at Pinna and at Oswestry were so bound down with bandages that less skin was exposed for heliotherapy than was exposed in his patients in plaster spicas; it was a different matter with spinal cases, and the cases that Dr. Kent Hughes had referred to were spinal. It would be of interest for him to state that he had seen no fewer than ten patients with tuberculous hip disease at Alton four years earlier aspirated for abscesses during the one morning.

Correspondence.

"ENSOL" IN THE TREATMENT OF CANCER.

SIR: Dr. E. H. Molesworth's letter published in THE MEDICAL JOURNAL OF AUSTRALIA (October 17, 1936) provides interesting reading, but calls for correction of certain misstatements and wrong inferences.

Firstly, Dr. Molesworth fails to quote the statements of the Commonwealth Minister for Health, the Honourable W. M. Hughes, in *The Sydney Morning Herald* of October 9, wherein Mr. Hughes asserted in the House of Representatives that his remarks anent deep X ray therapy of cancer had been made without any reference to any medical man.

Secondly, Dr. Molesworth inquires of my knowledge of X radiation. My experience of deep X ray therapy is limited to my observation on those cases of carcinoma and sarcoma from my own practice that have been treated with deep X ray by Dr. Molesworth and his colleagues. These cases had been operated upon by recognized surgeons, who performed wide radical resections, followed by a prolonged course of X radiation. At the conclusion of this combined treatment the patients have been returned to me with a letter of thanks and an expression of optimism for the future of such patients. Months later there has been recurrence of the growth locally and (or) metastases in remote areas, as the vertebral column, long and flat bones, liver, mediastinum and distant lymphatic glands. It has been my duty to sign the death certificate of a large proportion of these patients.

The spread of cancer had occurred in these cases after deep X ray therapy. It is *post hoc*. It can never be proved that it was *propter hoc*. This is the extent of my knowledge of the value of irradiation. Doubtless many members of the medical profession have had similar experience.

Cancer mortality statistics during the past decade must call for serious thought. In the appended table are quoted the figures for the largest cities in America, because in these cities are first-class hospitals, staffed with the best medical men and finest equipment to combat cancer.

This has been a period when deep X ray therapy has been widely used. The figures tell their own tale as to its influence. In England and Wales for the five-year period 1929-1933 the rate was 148·0 per 100,000 of population.

Thirdly, Dr. Molesworth speaks of "Ensol" therapy and that I am so optimistic as to its results. I have never been quoted as saying other than that it was a promising method.

Cancer in Selected American Cities, 1925-1935.

Year.	Number of Cities.	Population.	Deaths.	Rate per 100,000 Population.
1925	119	33,342,049	35,770	107·3
1926	132	35,422,353	38,864	109·7
1927	143	37,295,675	41,121	110·3
1928	146	37,996,870	43,426	114·4
1929	146	38,679,620	44,262	114·4
1930	177	41,440,122	48,648	117·4
1931	177	42,201,584	49,318	116·9
1932	177	43,005,389	50,347	117·1
1933	177	43,748,862	51,759	118·3
1934	184	44,840,442	55,201	123·1
1935	184	45,637,671	57,309	125·6

The patients that have been treated with "Ensol" in over 90% of cases have been those discarded by surgeons and deep X ray therapists. They had been sent home to die. "Ensol" has prolonged the life of 53% of such people. It has improved their general health, increased their weight, relieved 95% of such patients of their pain, restored many to their work, diminished the size of the growths, both locally and metastatically (growths have entirely disappeared in a large proportion of the cases). Surely it is a method of treatment to be persevered with and encouraged. It cannot be said that it has caused the sacrifice of a single life; rather has it extended life. "Ensol" treatment does not cause the patient pain or local or constitutional disturbance. In cases where "Ensol" has been used and the disease has not been so advanced, in Canada, America and Australia, surgical and radiation treatment has been advised concurrently.

Progress in medicine has always been achieved despite the opposition of those who sit in high places. I quote from the memoirs of Sir Almeric Fitzroy, Clerk of the Privy Council (June 25, 1907), with regard to the introduction of radium irradiation:

> I have been obtaining information for the Prime Minister on the subject of a Royal Commission on Cancer. Lords Lister and Rayleigh were the obvious people to appeal to, though in the present state of medical opinion, nothing but a dubious judgment could be expected. Lord Lister wrote very courteously, but expressed nothing more than the opinion of the medical authorities, which I know would be unfavourable to investigation of any independent body. Lord Rayleigh disclaimed any qualification to speak on his own responsibility but undertook to ascertain whether the Council of the Royal Society would advise on the point. The medical profession will hear nothing but the surgical treatment of cancer, partly because of its traditional repugnance to the introduction of new methods and possibly owing to the enormous profits that accrue from operations for cancer.
>
> Jenner, Simpson, and to a lesser degree Pasteur and Lister, have all in turn had to encounter the opposition of the mandarins of the profession.

Treatment of cancer with "Ensol" is being carried out in Sydney and will be continued. The final arbiters of its efficacy will be the patients, their relatives and medical men in general practice who attend cancer sufferers in their last agonies.

It is not my intention to enter into any controversy with Dr. Molesworth or any of his colleagues on the question of irradiation *versus* "Ensol" from any angle. I have never attacked surgical or biophysical methods of treatment of cancer, nor do I rush to the defence of "Ensol". Time will prove its value without advocates good or bad, strong or weak.

Yours, etc.,

NEVILLE DAVIS.

"Craignish",
185, Macquarie Street,
Sydney,
October 17, 1936.

Books Received.

STARLING'S PRINCIPLES OF HUMAN PHYSIOLOGY, edited and revised by C. Lovatt Evans, D.Sc., F.R.C.P., F.R.S., LL.D., and H. Hartridge, M.A., M.D., Sc.D., F.R.S.; Seventh Edition; 1936. London: J. and A. Churchill Limited. Royal 8vo, pp. 1,109, with 554 illustrations. Price: 24s. net.

A DIABETIC MANUAL FOR PRACTITIONERS AND PATIENTS, by E. L. Bortz, A.B., M.D., F.A.C.P., with a foreword by G. M. Piersol, B.S., M.D., F.A.C.P.; 1936. Philadelphia: F. A. Davis and Company. Demy 8vo, pp. 222, with illustrations.

RECENT ADVANCES IN RADIOLOGY, By P. Kerley, M.D., B.Ch. (N.U.I.), D.M.R.E.; Second Edition; 1936. London: J. and A. Churchill Limited. Demy 8vo, pp. 332, with 176 illustrations. Price: 15s. net.

LABORATORY PRACTICE (MANUAL OF PUBLIC HEALTH), by J. R. Currie and Contributors; 1936. Edinburgh: E. and S. Livingstone. Medium 8vo, pp. 397, with illustrations. Price: 21s. net.

A TREATISE ON MATERIA MEDICA AND THERAPEUTICS, INCLUDING PHARMACY, DISPENSING, PHARMACOLOGY AND ADMINISTRATION OF DRUGS, by R. Ghosh; Fourteenth Edition by B. N. Ghosh, F.R.F.P. and S.; 1936. Calcutta: Hilton and Company. Demy 8vo, pp. 789, with illustrations. Price: 12s. 6d.

THE PRINCIPLES OF TREATMENT FOR DIABETIC PATIENTS, by H. B. Ewen, M.B., Ch.B.; Third Edition, revised and enlarged; 1936. Wellington: A. H. and A. W. Reed. Crown 8vo, pp. 45. Price: 3s. 6d. net.

LEAGUE OF NATIONS PUBLICATIONS: QUARTERLY BULLETIN OF THE HEALTH ORGANIZATION; VOLUME V, NO. 2: THE FLY PROBLEM IN RURAL HYGIENE; 1936. Geneva: League of Nations Publications Department. Medium 8vo, pp. 156, with illustrations. Price:

Diary for the Month.

OCT. 27.—New South Wales Branch, B.M.A.: Medical Politics Committee.
OCT. 28.—Victorian Branch, B.M.A.: Council.
OCT. 29.—South Australian Branch, B.M.A.: Branch.
OCT. 29.—New South Wales Branch, B.M.A.: Branch.
NOV. 2.—New South Wales Branch, B.M.A.: Organization and Science Committee.
NOV. 3.—Tasmanian Branch, B.M.A.: Council.
NOV. 4.—Western Australian Branch, B.M.A.: Council.
NOV. 4.—Victorian Branch, B.M.A.: Branch.
NOV. 5.—South Australian Branch, B.M.A.: Council.
NOV. 6.—Queensland Branch, B.M.A.: Branch.
NOV. 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
NOV. 10.—Tasmanian Branch, B.M.A.: Branch.
NOV. 13.—Queensland Branch, B.M.A.: Council
NOV. 17.—Tasmanian Branch, B.M.A.: Council.
NOV. 17.—New South Wales Branch, B.M.A.: Ethics Committee.
NOV. 18.—Western Australian Branch, B.M.A.: Branch.
NOV. 19.—New South Wales Branch, B.M.A.: Clinical Meeting.
NOV. 24.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xviii, xix. and xx.

CHARTERS TOWERS DISTRICT HOSPITAL, QUEENSLAND: Resident Medical Officers.
DEPARTMENT OF DEFENCE, MELBOURNE, VICTORIA: Medical Officer.
GOVERNMENT OF TONGA: Medical Officer.
LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer.
MENTAL HOSPITAL, NEW NORFOLK, TASMANIA: Junior Medical Officer.
PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Resident Registrars.
ROYAL PRINCE ALFRED HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Officers.
THE PUBLIC SERVICE BOARD, SYDNEY, NEW SOUTH WALES: Medical Officers.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY Hospital are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

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